

CHAPTER: 6

SURGICAL INFECTIONS

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Surgical Infections

Pathogenesis

3 elements are important for surgical infections to take place:

- Infective agents.
- Susceptible host.
- A closed local space which is suitable for infection to occur.

A. Infective agents

- The development of infection is favored by the number and the virulence of the invading organisms.
- Involved organisms may be:
 - Aerobic:** as streptococci, staphylococci, klebsiella and E. coli.
 - Anaerobic:** as Bacteroides and Clostridium.
- Virulent organisms are highly invasive and cause primary infections.
- Infective organisms may be:
 - Endogenous** from within the body, as in gut perforation producing peritonitis.
 - Exogenous** (hospital acquired → called secondary or nosocomial infection).

B. Susceptible host

- The host response to bacterial invasion can be reduced by several conditions as:
 - Malnutrition.
 - Metabolic disease (D.M. and Uremia).
 - Immune suppression e.g. steroids, chemotherapy, radiotherapy and AIDS.

C. Closed suitable space

- Most surgical infections start in a susceptible poorly vascularized tissue such as a wound or a natural space with narrow outlet which is prone to obstruction as gall bladder and vermiform appendix.
- The risk of infection in wound is increased by:
 - Poor surgical manipulation with devitalization of the tissues → leaving a large dead space with hematoma formation.
 - Bad general condition (e.g. D.M., senility).
 - Bad hygiene.
 - Foreign body.
 - Ischemia.

Scheme for infection:

- Definition.**
- Etiology:** causative org., PDF, route.
- Pathology.**
- C/P:** symptoms: general & local
Signs: general & local.
- Complications.**
- Investigations.**
- Treatment:** medical & surgical.

Secondary infection

- It is an infection that occurs during or after treatment of another pre-existing infection. It may result from the treatment itself or from changes in the immune system.
- e.g.: Bacterial pneumonia after a viral upper respiratory infection is another example.

Primary infection:

- It is an infection that occurs due to first time of exposed to a pathogen. During which, the body has no innate defenses against the organism, such as antibodies.

Acute non-specific surgical infections

- Acute abscess.
- Cellulitis.
- Erysipelas.
- Boils.
- Carbuncle.
- Acute lymphangitis.
- Acute lymphadenitis.
- Hydradenitis suppurativa.
- Ludwig's angina.
- Surgical site infection.
- Blood infection (bacteremia & septicemia).

1- Acute Abscess

① Definition

- A localized suppurative inflammation.

* ② Etiology

Organism

- Usually **staph** (coagulase +ve), especially in skin & neck abscess.
- Less commonly **strep**, **E. coli**.
- The commonest organism in abdominal abscess is **E. coli**.

Predisposing factors

- Poor general resistance, **DM**, senility.
- Bad hygiene.

Route of infection

- Direct** → through a wound or ulcer.
- Blood** → from a septic focus (bacteremia).
- Lymphatic** → from an infected area.
- Natural passages** e.g. airways: lung abscess.

③ Pathology

The abscess formed of 3 zones.

1. Central zone → Pus

- Liquefaction, by dead WBCs.
- Pus is formed of: **necrotic tissue**, **inflammatory exudates**, **organism** and **dead leucocytes**.

2. Intermediate zone

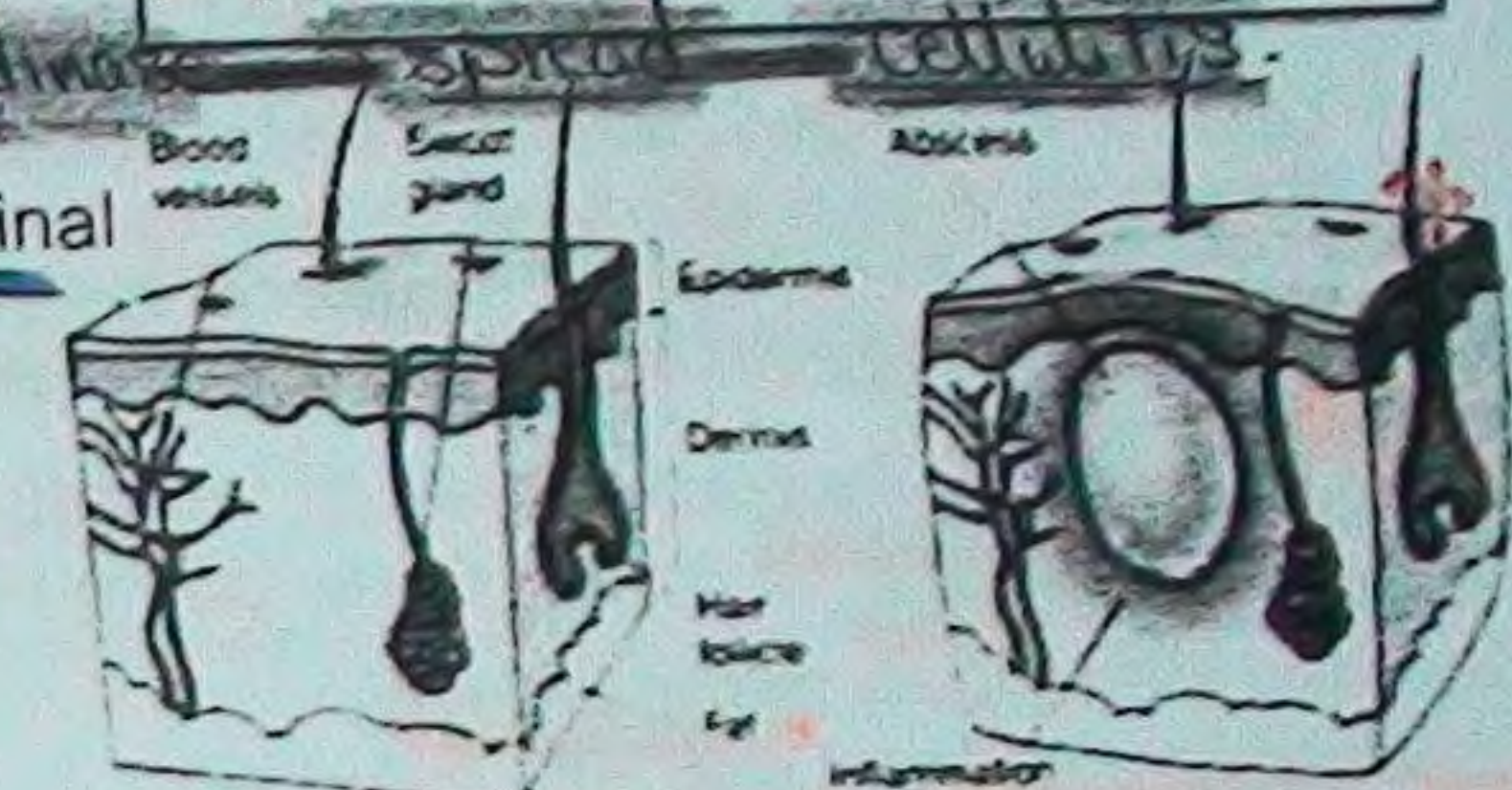
- Formed of **granulation tissue**.
- Appear to secrete pus, hence the old term (**pyogenic membrane**).

3. Peripheral zone

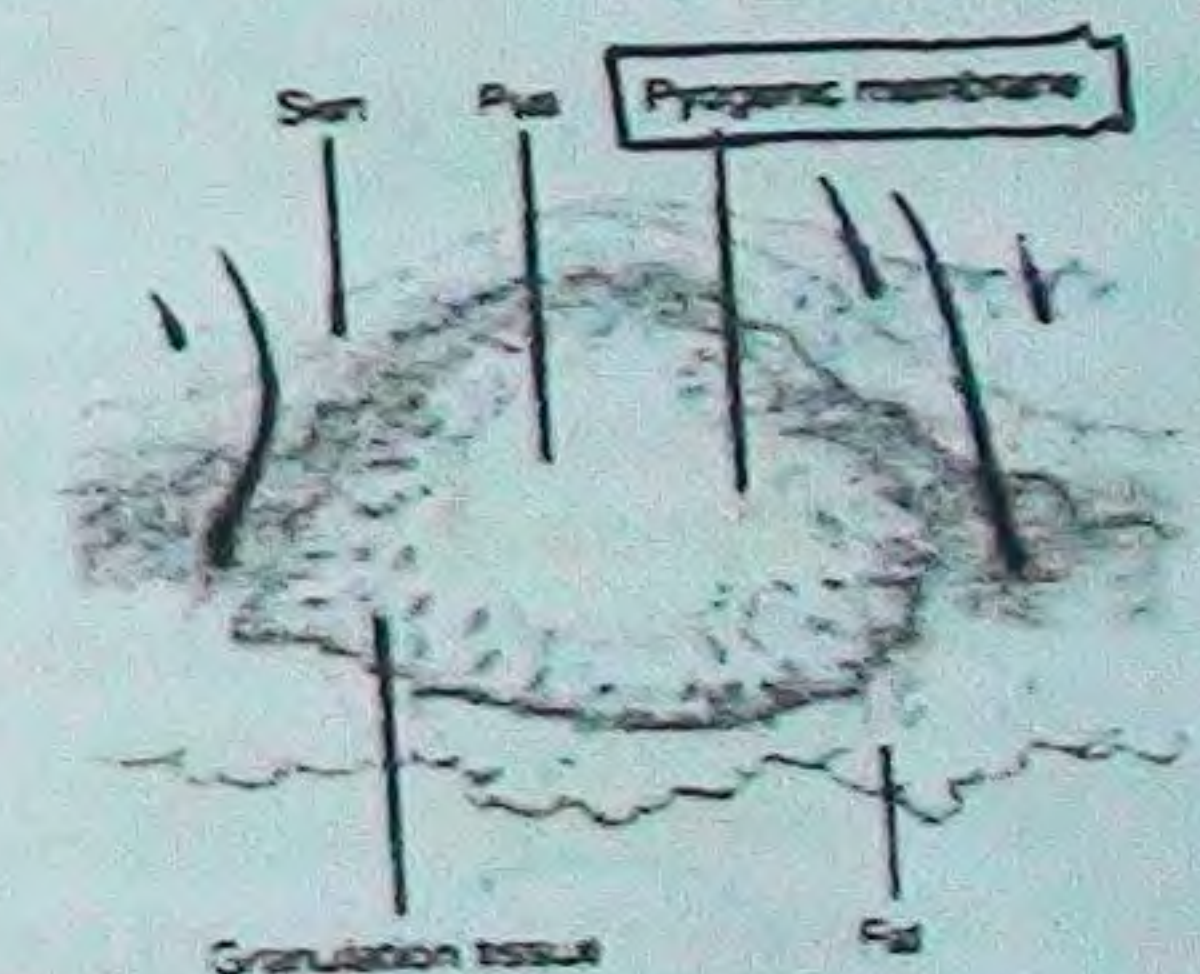
- Of **hyperemia**. **"Congestion"**

granulation tissue ← Pus

- Etiology:** -Skin: staph. Aureus
-Abdomen: E. coli
- C/P:** FAHM, pain, swelling, LN
- Comp.:** general, local
- Invest.:** CBC, C&S, acc. to site
- TTT:** AAA + incision & drainage



The Best Cover for a wound is **skin**



Microbe
cytokines
↓
PMN
تطاع بقى
ينفجر بالدم

Clinical Picture

Symptoms

⇒ **General:** (FAHM)...etc.

⇒ **Local:**

- **Pain:**

- At first dull aching pain before pus collection then → throbbing with collection of pus.
- Decreased by elevation of the part.

- **Swelling:** → due to edema of the tissues.

- **Loss of function** → due to pain.

Signs

⇒ **General:** fever (hectic or spiky), tachycardia.

⇒ **Local:**

- **Swelling** → Hot & red, Tender.
→ At first indurated then soften at center.
→ After 2-5 days pus points & ruptures.

- **LN's** → Enlarged, Elastic, Tender, Mobile.

* **Fluctuation** is a late sign

- An abscess starts as painful tender mass covered by skin, enlarged L.N. & FAHM.

- Once **pus** formed:

1. Pain become throbbing.
2. Fever become hectic.
3. Covering skin shows pitting edema.
4. Shooting leucocytosis with shift to the left.
5. Fluctuation:

6. no response to Treatment.

- Don't wait for fluctuation specially in the breast, prostate, parotid, penneum, pulp space (5Ps) & Ludwig's angina.
- Fluctuation is elicited easily in superficial abscess.
- False sense of fluctuation in breast abscess.

Examples of special clinical picture according to site

- Brain abscess: clinical picture of increased ICT.
- Lung abscess: pus on postural drainage.

Complications & Fate

General → Acute infection

- **Toxemia** → absorption of bacterial toxins into the circulation.
- **Bacteremia** → transient presence of bacteria into the blood. It may be dangerous in those with cardiac or orthopedic prosthesis → prophylactic antibiotics before minor procedures.

- **Septicemia** → persistent bacteremia multiplying in the blood & toxins.

- **Pyemia** → septic emboli released from an infected thrombus.

Local From septic thrombophlebitis.

1. **Pointing & rupture (Commonest sequel)**

- The pus passes along the line of the least resistance.
- It points on the skin, mucous membrane or serous surface.
- It ruptures & discharges its contents.

2. **Chronicity** due to: (antibioma) → chronic abscess Due to antibiotic.

- Organism tends to be chronic e.g. TB.
- Persistence of predisposing factors
- Inadequate drainage & treatment.

↓ Congestion ↑ Fibrosis

remission & exacerbation with fluctuation of immunity.

3. Spread:

- Cellulitis.

- Lymphangitis.

- Lymphadenitis.

4. **Sinus** (epithelialized tract connected to skin surface) & **Fistula** (pathological tract between 2 epithelial surfaces) formation, especially with actinomycosis and mycobacterium.

5. Antiboma:

- It is a subtype of chronic abscess that occur when antibiotics is given in acute abscess **without drainage**.
- It contains sterile pus.
- Occurs commonly in breast.
- It is misnomer as it is an abscess not a tumor.

6. Investigations

- **CBC:** leucocytosis.
- **Culture and sensitivity.**
- **Plain X-ray** e.g. lung abscess.
- **U/S** e.g. amoebic liver abscess.
- **CT and MRI** e.g. brain abscess.
- **Aspiration** can be done guided by X-ray, U/S, CT or MRI.

7. Treatment

General

- Analgesics, antipyretic and antibiotic (even parenteral in severe cases) (AAA).
- Antibiotics are **not** alternative to drainage, but given when infection is not under control and spreading.

Local

1. Rest of the affected organ + TTT of predisposing factors.
2. Hot fomentation (hyperemia brings more antibiotics & antibodies).
3. **Incision & drainage:**

- Under general anesthesia or local regional anesthesia.
- The incision should be:

→ Long & Dependent → allow good drainage.

→ Never cross a skin crease.

→ Parallel to important structures e.g. nerves & vessels.

- After incision → introduce a finger (or mosquito) to break all septa.

- Packing for 48 hours for hemostasis.

- Later on → dressing **without** packing every day.

- After pus discharge becomes minimal → dressing every few days until complete healing.

Hilton's methods

- In areas with important structures (e.g. axilla, neck, parotid).

- The skin is only incised.

- Abscess cavity is entered by closed blades of artery forceps.

- Then the blades are opened widely.

Aspiration used in

- Amoebic liver abscess
- Brain abscess
- Cold abscess

Chronic abscess:

- Thin walled → incision & drainage
- Thick walled → excision

Key points

1. Antiboma: not a tumor and common in the breast.
2. Abscess which we don't wait for fluctuation (5Ps).
3. How to drain abscess and Hilton technique.

Penicillin is not effective against (staph) being coagulase penicillinase +ve
abscess/boil → augmentin. ampicillin + Glutamic acid.

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General Surgery

2- Cellulitis

Definition

- Is an invasive non-suppurative infection of loose C.T. (although PMNL are abundant, there is no pus formation).

Etiology

Organism

- Usually strept → produces hyaluronidase & fibrinolysin → spread.
- Sometimes staph → into the superficial skin structures.

Predisposing Factors & Route

- Poor general resistance.
- Bad hygiene.
- Scratch or prick.

Clinical Picture

Deep

Symptoms

See before

Signs

⇒ General: see before.

⇒ Local

- Tender, dusky red, hot area with indurated edge (never affect ear pinna)
- LN's → enlarged, elastic, tender, mobile.
- Red streaks of lymphangitis (d.t. lymphatic spread).
- Spreads rapidly with ill defined edge.
- No suppuration.

Complications

- General: bacteremia, septicemia, toxemia & pyemia.
- Others: chronicity + post-streptococcal glomerulonephritis + scarlet fever + SIRS (common).

DD

- Contact allergy.
- DVT
- Chemical inflammation (at the site of drug injection).

Investigations

- CBC: leukocytosis.
- Blood cultures are often negative.

Treatment

- Rest in elevation of affected part & hot packs.
- Antibiotic of choice is penicillin (benzyl penicillin 600-1200 mg i.v./6hours). Erythromycin is used as alternative in patients allergic to penicillin.
- If no response after 48 hrs → either diagnosis of cellulitis is doubted & an abscess is suspected or resistant organisms are involved.

Key points

- Non-suppurative and poorly localized.
- SIRS is common + GN and scarlet fever.
- Antibiotic of choice is penicillin.

واحد عنده Cellulitis
في وشه عدين فيم روح الودين
Erysipelas
Erysipelas
(subcutaneous fat)



staph is penicillinase +ve
positive

Sore Throat → Streptococci → RF → 100% Penicillin
Skin infection → GN → 100% Penicillin

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3- Erysipelas

Definition

- Non suppurative diffuse sharply demarcated streptococcal infection of lymphatics of the skin.

Etiology

Organism

- Usually strept pyogenes (hemolytic strept)

Predisposing Factors & Route

- Poor general resistance, DM & senility.
- Bad hygiene.
- Minute scratches or abrasions.

Clinical picture

- Symptoms of toxemia are present.
- Locally the picture is similar to cellulitis but with some differences:
 - The skin is rose pink.
 - The edge is well defined, slightly raised and often shows minute vesicles just beyond the spreading margin.
 - There may be islets of inflammation beyond the spreading margin separated from the main area by apparently normal skin.

Complications

- Facial erysipelas may lead to cavernous sinus thrombosis.
- Septicemia.
- Recurrent erysipelas may block the lymphatics leading to elephantiasis.

DD 1. Contact allergy.

2. Herpes Zoster.

Investigations

- Swabbing for culture and sensitivity.

Treatment

- Isolation of the patient as the disease is highly contagious.
- Rest in elevation of affected part.
- Hot packs.
- Antibiotics.

4- Boil (Furuncle)

Definition

- Acute staphylococcal infection of hair follicles or sebaceous gland → perifolliculitis (deep infection).

Etiology

Organism

- Usually Staph aureus → produce necrotoxin, responsible for the necrosis & sloughing.

Predisposing Factors & Route

- Poor general resistance.
- Bad hygiene.

- Etiology: staph. aureus + DM
- C/P: FAHM, pain, swelling, LN
- Comp.: as abscess + CS thrombosis
- Invest.: blood sugar for DM
- TTT: AAA + cause + comp.



Pathology

Sites

- Hairy areas (common in face, neck & axilla) **not** in palm, sole.

Pathogenesis

- Perifolliculitis - central necrosis & suppuration.
- Surrounding area becomes red, hot, tender & a hair is coming out from it.

Clinical picture

Symptoms

- See before.

Signs

General:

- FAHM, marked constitutional symptoms are common.

Local:

- Swelling → Hot & red, tender → at first indurated then soften at center → after 2-3 days → pus points & rupture.
- LN's → enlarged, elastic, tender, mobile.

Complications

- General + Chronicity & Spread
- Cavernous sinus thrombosis:** if manipulated or squeezed in the dangerous area of the face (the upper lip, septum of nose & adjacent area) → spread via pterygoid plexus or ophthalmic veins.
- Blind boil:** Boil subsides leaving a thick indurated area.

Investigations

- Blood sugar for recurrent and severe cases.

Treatment

- See before (AAA + rest + hot fomentations) +
- Improve general condition e.g. vitamins, control DM, good nutrition.
- Antibiotic of choice are penicillinase resistant antibiotic as flucloxacillin (systemic antibiotics are often required).

Key points

- Infection around hair follicle.
- Complications: cavernous sinus thrombosis and blind boil.
- Treatment: blood sugar control + improve general condition.

5-Carbuncle (Infective gangrene of the nape) ✓

Definition

- Acute staphylococcal infection of S.C. tissues → infective gangrene

Etiology

Organism

- Usually **Staph aureus** → produce potent **necrotoxin** responsible for the necrosis of tissues & necrosis of blood vessels due to sloughing.

- Etiology:** staph. aureus + DM
- C/P:** FAHM, pain, swelling, LN
- Comp.:** as abscess + CS thrombosis
- Invest.:** blood sugar for DM
- TTT:** AAA + cause + comp. + debridement

Augmentin
penicillin
Glycolonic acid
عاشان يورط
ال penicillinase

Predisposing Factors & Route

- See before
- Diabetics are more common.

Pathology

Sites

- Nape of neck (commonest), back, face & dorsum of hand.

Pathogenesis

- Infection starting in hair follicles in skin.
- Then, Spreads to underlying fatty SC tissue → separate loculated abscesses → each abscess burst on the surface by a separate opening (i.e. a single carbuncle has multiple openings).

Clinical Picture

- There is usually severe toxemia.
- Usually starts as painful induration of the skin and subcutaneous tissue.
- The skin is red.
- As the swelling enlarges its central part become soft but usually no fluctuation can be detected.
- Multiple areas of skin thin out and separate forming multiple sinuses through which multiple sloughs separate slowly.
- Usually no frank pus is formed.

Complications

- General + Chronicity & Spread
- Cavernous sinus thrombosis** if manipulated or squeezed in the dangerous area of the face (the upper lip, septum of nose & adjacent area) → Spread via pterygoid plexus or ophthalmic veins.

Investigations

- Blood sugar for recurrent or severe cases.

Treatment

- See before (AAA + rest + hot fomentations) +
- Antibiotic of choice is penicillinase resistant as flucloxacillin.
- Improve general condition e.g. vitamins, control DM, good nutrition.
- Once pus is formed → **cruciate incision** opening of all pockets.
- Debridement** with removal of all the necrotic tissue.
- Dressing** until healthy tissue is formed (culture is -ve).
- Leave to heal or use skin graft.
- Glycerine Mg sulfate** may be applied until sloughing occurs.

Key points

- Infection starts in the hair follicle.
- Commonest site is nape of the neck.
- May be complicated by cavernous sinus thrombosis if manipulated or squeezed in dangerous area of the face.
- How to drain.



الغلا حبة
الغلا حبة

تسبب لونه اصفر
وتغير لونه لحد ما يمتلئ
لونه اصفر
تصلب خطي
اللدوخ

لوني تسبب جرح حاد
السكري في الشارب اقله

اول ما يمتلئ
اقله
Skin Graft
Skin Flap

اجمر ونقطة
جنترا

ري البريل
نسل بواق
Necrotic
Tissue

6- Acute Lymphangitis

Definition

- It is infection of lymph vessels by organisms usually streptococci

Route of infection

- Entering through an abrasion or a wound in area drained by lymphatics

Clinical presentation

- If main trunk is affected → red streaks in the overlying skin (tubular lymphangitis).
- Fever & rigors usually present

Treatment

- Systemic antibiotics + hot applications.

7- Acute Lymphadenitis

Definition

- It is enlarged infected lymph nodes.

Route of infection

- Spread of infection along lymphatics from a septic focus in drainage area.

Clinical presentation

Symptoms

- General : FAHM
- Local
 - Pain, swollen & tender lymph nodes.

Signs

- Toxemia usually present (e.g. fever & tachycardia).
- Primary focus of infection may be detected to the draining area.

Treatment

- Systemic antibiotics + hot application.
- If suppuration is formed incision & drainage is essential.

8- Hydradenitis suppurativa

- Mixed staph. & strept. infection of apocrine sweat gland in perineum or axilla producing multiple abscesses & pus discharging sinuses.
- Infection is difficult to eradicate & can progress to chronicity with considerable reaction in perineum.

- Multiple anal fistulae (differentiated by absence of internal openings).

Treatment

- Drainage of abscess followed by careful hygiene, painting with disinfectants & antifungal
- Or
- Excision of apocrine sweat glands followed by skin graft.

9-Ludwig's Angina (Deep Cellulitis of the Neck)

- It is a surgical emergency.

Definition

- Bilateral diffuse cellulitis of the floor of the mouth & pharynx.
- Floor of the mouth is a CT-space divided by myelohyoid muscle into submandibular and sublingual spaces.

Etiology

Causative organism

- Mainly mouth commensals (strept).

Route of infection

- Direct → from teeth (dental caries) → or submandibular sialadenitis.
- Ulcer of the tongue.

Clinical Picture

Symptoms

- General:
 - Fever, anorexia, headache, malaise
- Local:
 - Severe dysphagia and severe dyspnea as the tongue is pushed upward and backward → may obstruct food and air passages.
 - Suffocation due to laryngeal edema or obstruction by the tongue.

Signs

- General:
 - Fever.
- Local:
 - Pharyngeal: edema of the floor of the mouth, the tongue is pushed upward and backwards.
 - Cervical: swelling in submandibular region.

Treatment

- Medical:
 - Massive doses of antibiotics. (early by penicillin)
 - Bed rest in semi-sitting position to avoid airway obstruction.
- Surgical:
 - Submental curved incision including the skin & deep fascia (there is no or little pus because suppuration seldom occurs).
 - Tracheostomy when necessary. Resp. Distress.

Cellulitis of the neck

- Superficial cellulitis: common & treated as cellulitis anywhere else, rarely if above the level of the hyoid bone can lead to glottic edema & asphyxia in children.
- Deep cellulitis (Ludwig's angina)

in Floor of the Mouth
(Digastric Δ)



حالة طرية خطيرة

التهاب في اللغز
بتروح الحزاز وتقول
هات
اللسان
واللغز

التهاب في الغدد
تطلع مياة

تتميز بأن
الحشوات وحشوة

كل مشوة يجب له حشوة
Acute exacerbation of chronic

بيديتي
edema
على فوهة اللسان
أحتمل في
Deep Fascia

Key points

1. Infection of the floor of the mouth.
2. Clinical picture of dyspnea & dysphagia + pharyngeal & cervical examination.
3. Sub-mental release incision including the skin and deep fascia.
4. Tracheostomy may be indicated.

* 10- Surgical Site Infection (SSI) (Surgical Wound Infection)

hospital acquired infection

Definition

- They are infections of tissue, organs & spaces exposed during the performance of invasive surgical procedures.
- They are classified into:

- Etiology:** single or multiple organism P.F.=Bad (wound, patient, surgery)
- C/P:** FAHM, pain, swelling, LN
- Comp.:** as abscess + gangrene, SIRS, MOF
- TTT:** -prophylactic: acc. to class
-curative: AAA + drainage

a) **Incisional** → they are either superficial limited to skin & S.C. tissue

→ Deep in musculoaponeurotic layers.

b) **Organs:** e.g. lung (hospital acquired pneumonia).

c) **Spaces:** → e.g. subphrenic or pelvic abscesses "space infection"

Causative Organisms

- The most common causative organisms associated with wound infections include Staphylococcus aureus (MRSA), Streptococcus pyogenes, Enterococci and Pseudomonas aeruginosa
- Synergy of gram -ve bacilli and anaerobic bacteroids (abscess after abdominal surgery).

- Direct
- Blood
- Lymph.

Source of Infection

- > **Endogenous:** human body flora.
- > **Exogenous:** from surgical team, instruments, dressing etc....

Predisposing Factors

(Most important factor is nature of the surgery)

General patient factors Vit A, C Zinc	- Old age, obesity, malnutrition - Metabolic disorders - Immunosuppression as DM, uremia & malignancy. - Drugs ↓ immunity as steroids & chemotherapy. - Anemia
Wound factors	- Nonviable tissue in wound - Tissue ischemia - Hematoma formation - Foreign bodies
Operative factors	- Nature of surgery (classified into 4 classes) - Poor surgical technique - Long operation time (>2 hours) - Intra-operative contamination - Prolonged preoperative stay - Presence of foreign bodies or prosthetic material. - Poor skin preparation.

* **Dead space:** a space remaining in the tissues as a result of failure of proper closure of surgical or other wounds, permitting the accumulation of blood or serum.

ليطلع مادة من
organism
أفر

حاجو

Classification

Classified according to load of bacteria at time of operation into:

A. Clean wounds (class I):

- Elective non-traumatic wounds in which the gastrointestinal, urinary or respiratory tracts are not entered.
- Risk of SSI → less than 2% → endogenous.

B. Clean contaminated (class II):

- Elective surgery into the gastrointestinal, urinary or respiratory tracts with no significant spillage.
- Risk of infection → 2-5%.

C. Contaminated wounds (class III):

- Open traumatic wounds encountered within 4 hours
- Gross spillage from GIT, e.g. gastrectomy when clamp is released.
- Incision through non-inflamed, non-purulent tissues.
- Risk of infection → 10-20%.

D. Dirty wounds (class IV):

- Traumatic wounds more than 4 hours
- Purulent infection or necrotizing soft tissue infection.
- Perforated viscus accompanied by high degree of contamination (e.g. perforated appendix).
- Risk of infection → up to 40%.

Clinical Picture

- Usually appears between the 5th & 10th day postoperative.
- Earliest manifestations are: wound pain & post-operative fever.

Symptoms

> **General**

- Fever, anorexia, headache & malaise.

> **Local**

- Pain.
- Swelling "sutures dipping through the skin".
- Disturbance of function: The wound hasn't healed within 10 days after the injury.

Signs

> **General:**

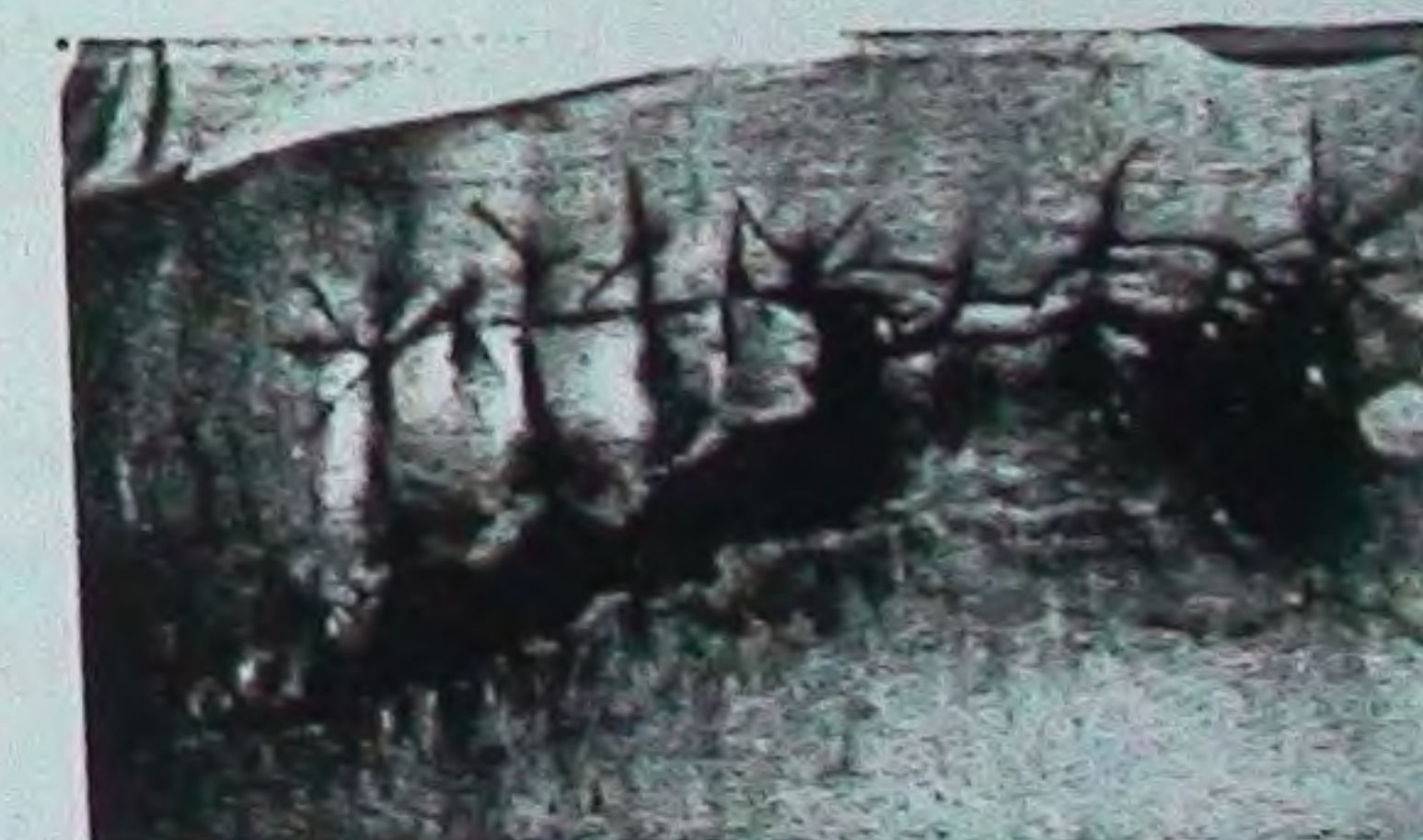
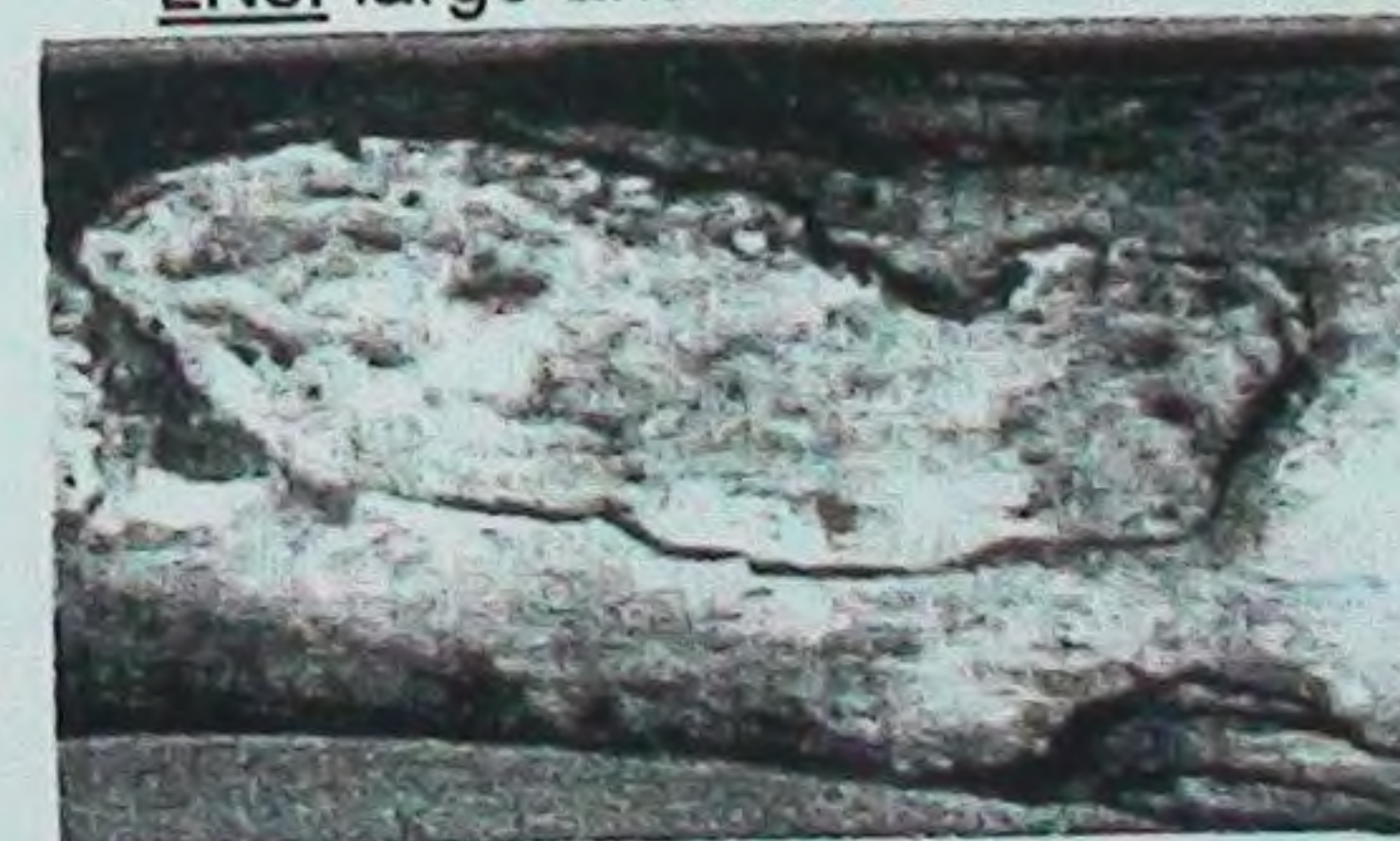
- Fever & tachycardia

> **Local:**

• **The wound:**

1. Show redness, tenderness, may ooze pus or cloudy fluid from the wound & yellow crust has formed on the wound.
2. Fluctuant areas and crepitus are occasionally felt

• **LN's:** large and tender.



Give prophylactic antibiotics

بالأدوية والعدوى
No prophylactic antibiotics

مؤقتة بس. بشكل عام في العدوى
infection بس بشكل عام في العدوى

here antibiotics are curative

MRSA
vancomycin
Resistant
Staph aureus

متردد في فحوصات
antibiotic
So → superselection

N.B.: Infections to the deep fascia may be difficult to recognize. They are associated with systemic signs of inflammation.

Recommendation to minimize surgical site infection

The patient

1. Correct any PDFs as control of diabetes, stopping of smoking and correction of nutritional deficiency.
2. Operations are avoided in patients with active infections if possible.
3. Shaving or clipping the hairs just before skin incision.
4. Skin preparation with appropriate antiseptics.

The surgeon

1. Surgeon should have short nails & should scrub properly.
2. Meticulous surgical technique by proper hemostasis, gentle handling of tissue & avoiding tight sutures or leaving dead space.
3. Delayed primary closure of heavily contaminated wounds.

Prophylactic antibiotics

- Evidence based surgical practice showed that in clean contaminated & contaminated wounds post-operative infections can be avoided by using appropriate prophylactic antibiotic.
- The choice of prophylactic antibiotics:
 - Select the antibiotics active against the organism likely to be encountered in the hospital.
 - Select the antibiotics which give high concentrations in the required tissues used for a suitable time.
 - Select the antibiotics which are unlikely to induce renal or hepatic damage if such damage already occurs, correct dosing regimens should be used.
- The evidence that newer expensive broad spectrum antibiotics are more effective for prophylaxis than the cheaper narrower spectrum antibiotics is weak.

Scheme to follow according to classification of wounds:

Clean operations:

No need for prophylactic antibiotics except in:

- When an implant, e.g. prolene mesh or a vascular graft is used.
- In patients with valvular heart disease to prevent infective endocarditis.
- In emergency surgery in a patient with pre-existing active infection.
- When infection would be very severe or have life-threatening consequences as in aortic surgery or organ transplantation.

Recommendation:

- One dose of 1st generation cephalosporin, or (ampicillin + sulbactam).

Clean contaminated operations:

- One dose of 2nd generation cephalosporine or (ampicillin + sulbactam) + aminoglycoside.

Contaminated operations:

- As clean-contaminated, but add metronidazole.

All operation exceeding 2 hrs, another dose of antibiotic is prescribed.

Treatment

- Liberal drainage. The wound should be opened by removal of skin stitches.
- Antibiotics are used in invasive infections guided by culture & sensitivity tests.
- Possible sources of hospital acquired infection should be traced and corrected.

→ No clia → atelectasis

هيفوف
ف
secretions

obesity
Risk of Devitalization
↑ Risk of infection

No sutures
under
Tension

تتاع الحذير
قبل ما يركب
انابيبه

بند
مراجعة الطاب
قبل العملية
بنفس ساعة

Quinolones
هيتاخذش في اقل من 4 18

لو
GIT
اتلقت
عننا الازع
Metronidazole

Anaerobic

Special Types of Hand Infection

Acute Paronychia 30%

→ The Commonest Hand infection

Definition

- Infection of the tissue surrounding the nail bed.

Etiology

Organism

Predisposing Factors & Route

- Commonest hand infection (30% of cases).
- Thorn is driven under nail.

Clinical Picture

Symptoms

- See before + swelling of nail fold.

Signs

⇒ **General:** see before.

⇒ **Local:**

- See before +
- Nail bed indurated.
- Later on become cystic, yellowish.
- At first at one side latter on U shaped around the nail fold.

Complications

- Trimming skin around nail Spread of infection under nail → subungual abscess.

Treatment

- See before +

Incision

- Oblique incision at the angle of the nail fold ± excision of the outer 1/4 of nail.
- U-Shaped incision → if pus present all around the nail.
- Excision of outer quarter of nail → if pus under the nail (subungual abscess).

Acute subungual infection

- Infection of the space between subungual epithelium & their periosteum usually due to prick beneath the nail.
- C/P: severe pain & little swelling. The maximum tenderness is beneath the free edge of the nail where pus comes out.
- III: removal of small V from the center of the free edge of the nail.

Key points

- Commonest hand infection (30%).
- May be complicated by subungual abscess.
- Specific clinical picture.
- Comp.: subungual abscess.
- Oblique incision.

at nail sulcus

آخر عقلة من ورا



وهي طلع تاني
خلشان انا
لايب الجدر، شاع
موجود في
Periosteum

epiphysis → epiphyseal Cartilage "physis"
metaphysis
Diacaphysis

Chronic paronychia

هو ضيق جلدي

لاكثر من اربعين ← فترت موانع

عسر القصب

(اديعي على طول في المياه)

Etiology

- Fungal infection (not related to acute paronychia).
- Patient whose hand is frequently emerged in water.

Clinical picture

- Itching and whitish nail bed.

Investigations

- Culture on Sabouraud's agar.

Treatment

- Keep the hand dry + topical antifungal cream.
- If this fails, the nail fold is laid open & nail extraction may be required.

Pulp Space Infection (Felon)

مهم جداً جداً

It is a surgical emergency "do not wait for fluctuation".

Anatomy

- It is the SC compartment which is related to the palmar surface of the distal phalanx.
- It contains:
 - Fat
 - Fibrous tissue septae (pus is trapped between it), which extend from the skin to periosteum.
 - Digital artery which gives epiphyseal branch before it enters the space
- It is separated from the distal volar space by a deep fascia.

end artery

Definition

- Infection in SC compartment related to the palmar surface of the distal phalanx.

Incidence

- The 2nd common hand infection.

Etiology

- Usually a prick

Organism

- See before.

Predisposing Factors & Route

- See before.

Pathology

- See before.

Clinical Picture

Symptoms

- See before + swelling of distal phalanges.

Signs

⇒ **General:** See before. Fever, Tachycardia

⇒ **Local**

- See before +
- The distal phalanx is first indurated.
- Later on become cystic, yellowish.



* **Complications**

- Thrombophlebitis of digital vessels → necrosis → infection (Osteomyelitis of the distal phalanx) → except epiphysis → Parrot peak deformity. *Sequestrum* *الغصنة* *ماتت*
- Septic arthritis. *التهاب المفاصل*

* **Treatment**

- See before +

Incision

- Early surgical drainage is indicated, the incision is sited directly over the most tender point. *الغدة مانت*
- Hookey stick → for sequestrectomy (The incision should be deepened to divide all septa).

Key points:

1. Anatomy of pulp space.
2. It is the 2nd common hand infection.
3. Specific clinical picture.
4. May be complicated by parrot peak deformity and septic arthritis.
5. How to incise.

Suppurative Tenosynovitis**1. Tenosynovitis of Middle 3 Fingers****Anatomy**

- Each finger of these has a separate synovial sheath.
- Proximal end:**
 - At the level of metacarpophalangeal joint.
- Distal end:** at base of distal phalanges.
- They enclose the flexor tendons.
- Dilated Cul- de sac.

Etiology**Organism**

- See before. *staph.*

Predisposing Factors & Route

- See before. *Direct*

Clinical Picture**Symptoms**

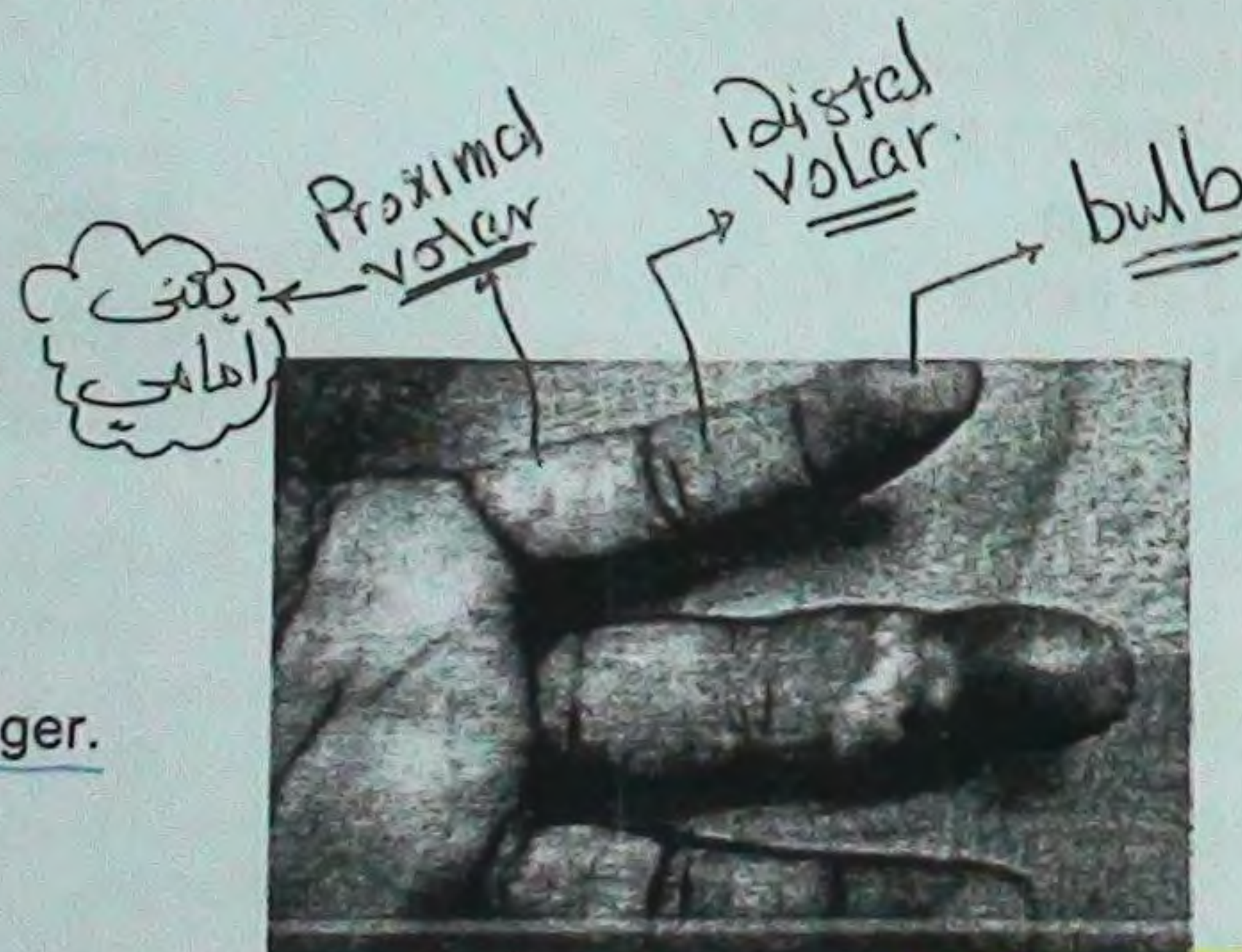
- See before + swelling all around the finger.

Signs

- General:** see before.

Local:

- See before + classic signs described by Kanaval. *(hookey stick)*
- The affected finger is semiflexed with limitation of movements → hook sign. *الغصنة*
- Symmetrical swelling of entire finger. *Mainly Prox & Distal volar.*
- Tenderness over the infected sheath specially its proximal "cul de sac" point

**Complications**

- Sloughing of the tendon due to interfering with nutrition of the tendon.
- Adhesions inside the synovial sheath → limitation of movement.
- Osteomyelitis
- Arthritis.

Treatment

- See before +

Incision

- Transverse over proximal cul de sac → the sheath bulges & is incised → introduce a urethra catheter → irrigate with penicillin.
- In severe cases → counter incision distally.

Key points:

1. Anatomy of synovial sheath of middle three fingers.
2. Specific clinical picture.
3. Hook sign.
4. How to incise.

2. Ulnar Bursitis**Anatomy**

- Larger than radial bursa.
- Distally connected with synovial sheath of little finger.
- Proximally → run between the flexor retinaculum and pronator quadratus → extend to the forearm (called space of Parona).
- Envelop the flexor tendons of the medial 4 fingers.

Etiology**Organism**

- Staph (80%), Strept, E. coli, G -ve bacilli.

Predisposing Factors & Route

1. Bad hygiene.
2. Bad general condition.
3. More in manual workers & housewives.

Clinical Picture**Symptoms**

- General** → FAHM.
- Local**

Pain → dull aching then become throbbing when pus is formed.
→ Decreased by elevation of the hand.

Swelling → according to site e.g. pulp space → distal phalanx, acute paronychia → nail fold, edema at the dorsum of the hand is common, irrespective of the site of infection.

Disturbance of function → limited movement.
Swelling of little finger, palm, distal part of forearm.

Signs**General**

- See Before.

Lateral

Radial

space of Parona

distal Bone. hook of hamate

ulnar

*Middle 3 fingers مش وارمين بس لو مركب هيو صوابشان
Their Tendons
Ulnar Bursa. معديين في*

Local:

- See before.
- Slight semiflexion of the little finger (hook sign).
- Limitation of movements of the medial 4 fingers.
- Edema of the dorsum.

MCQ

Kanavel's sign: tenderness over an infected ulnar bursa between transverse palmar creases & hypothenar muscles.

Complications

1. Sloughing of the tendon due to interfering with nutrition of the tendon.
2. Adhesions inside the synovial sheath → limitation of movement.
3. Osteomyelitis.
4. Arthritis.

Treatment**Before Suppuration**

General: antibiotics, analgesics, antipyretic. (AAA)

Local:

1. Hot fomentation.
2. Arm position → to diminish pain & edema.
 - Minor infection → arm to neck sling.
 - Major infection → elevated above level of body.
3. Hand position:

- If resolution is expected → **position of rest**:

⇒ Description of position: the patient grasps a ball of cotton with max Flexion of little finger, least flexion of index with the thumb in opposition.

Cloth ball



Position of rest

- If stiffness is expected → **position of function**:

- ⇒ The fingers are approximated to the thumb as if holding something.
- ⇒ Especially in tenosynovitis for fear of fibrosis of tendon & shortening → clawing of hand.

as if you were picking object.

After suppuration → Incision & drainage

1. Should be done once pus is formed.
2. Under general anesthesia, ring anesthesia for minor cases (better to be avoided).
3. Bloodless field by:
 - Elevated the limb for 2 minutes.
 - Sphygmomanometer cuff is inflated for 40 mmHg above systole.

4. Incision:

- Along radial border of hypothenar eminence

In severe cases → counter incision at the lower part of forearm

5. All pus is removed & granulation tissues are curtailed.

6. No drain is allowed because it causes pressure to delicate structures but a piece of tulle-grass might be allowed.

7. Dry dressing is employed; changed after the 1st day, then every 2 hours.

Key points:

1. Anatomy of ulnar bursa.
2. Specific clinical picture.
3. How to incise.

Longitudinal
incision
longitudinal
incisions
skin creases
over the
hypothenar

3. Radial Bursitis**Anatomy**

- Small in size.
- Distally connected with synovial sheath of the thumb.
- Proximally → run between the flexor retinaculum and pronator quadratus → extend to the forearm (called space of Parona).
- Envelops the tendon of flexor pollicis longus.

Etiology**Organism**

- See before.

Predisposing Factors & Route

- See before

Clinical Picture**Symptoms**

- See before + swelling of thumb, thenar eminence & distal part of forearm.

Signs

General: see before.

Local:

- See before + thumb is semiflexed.

Complications

- Sloughing of the tendon due to interfering with nutrition of the tendon.
- Adhesions inside the synovial sheath → limitation of movement.
- Osteomyelitis
- Arthritis.

Treatment

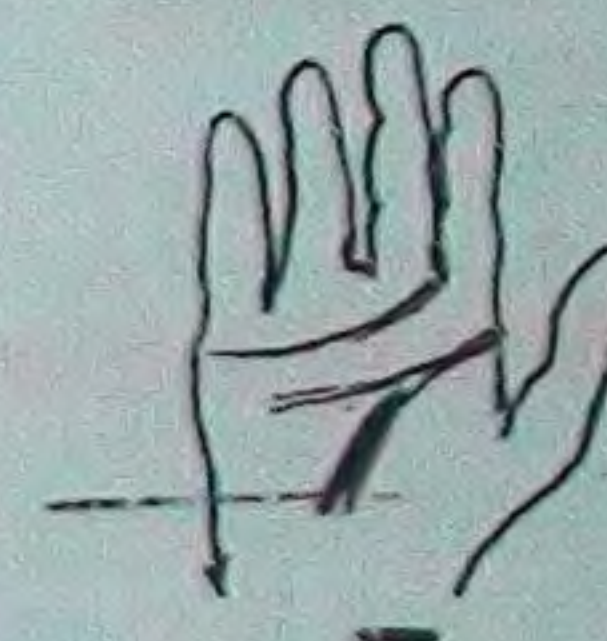
- See before +

Incision

- On the ulnar side of the thenar eminence stopping proximally 1.5 inches distal to the distal crease of the wrist to avoid injury of the motor branch of median nerve
- In severe cases → counter incision

Key points:

1. Anatomy of the radial bursa.
2. Specific clinical picture.
3. How to incise.



لازم أن يكون

→ (ape hand)

Loss of
opposition
thumb is in
the same
plane of other
fingers

Web Space Infection

Anatomy

- Triangular region between the dorsum & ventral skin present at the bases of the fingers.
- They contains: fat, digital vessels & nerves, lumbricals & Interossei.
- 1st & 2nd web spaces are connected to thenar space, while 3rd & 4th web spaces are connected to palmar space.

Etiology

Organism

- See before.

Predisposing factors & Route

- See before + Direct
- Extension from:
 - Adjacent web spaces.
 - Deep mid palmar space.

Clinical Picture

Symptoms

- See before + swelling of the web space.

Signs

- General:
 - See before.
- Local:
 - See before +
 - Swelling of the web space → Hot & red, Tender.
 - Sign of victory (the finger can not be approximated)

Complications

See before +

- Spread to deep mid palmar space & adjacent web space.

Treatment

See before +

Incision

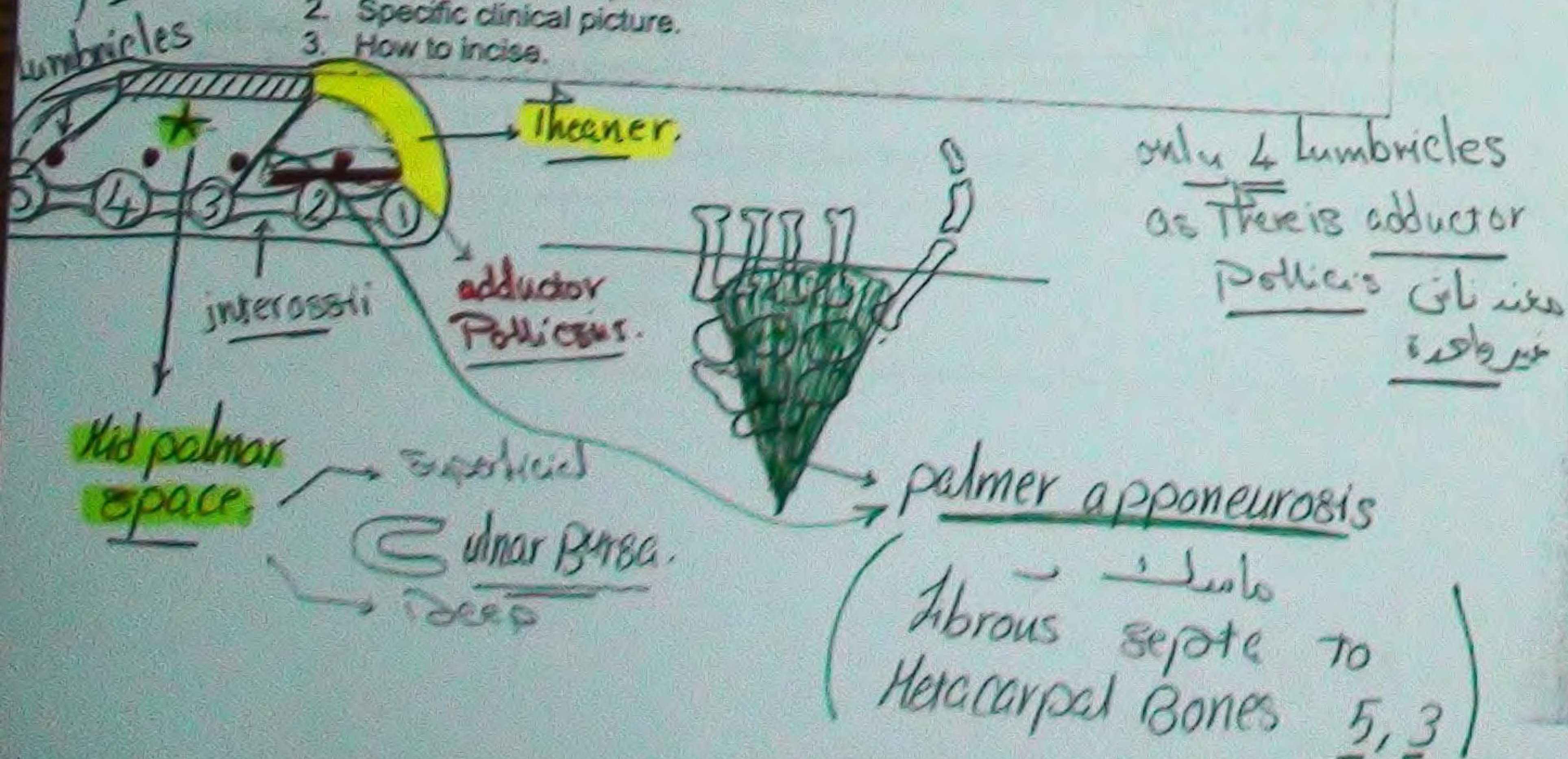
- A transverse incision over the web one cm from the free margin
- Counter incision posteriorly → severe cases

Free Border

to avoid fibrosis

Key points:

- Anatomy of web space.
- Specific clinical picture.
- How to incise.



Thenar Space Infection

Anatomy

- Ant → thenar muscle, radial bursa, and flexor pollicis longus.
- Post → adductor pollicis.
- Med → deep mid palmar space.
- Distal → extend to the web of the thumb.

Etiology

Organism

- See before.

Predisposing factors & Route

- See before.

Extension from:

- Tenosynovitis of the thumb.
- Deep mid palmar space.

Clinical Picture

Symptoms

- See before + swelling of the thenar eminence

Signs

- General: see before
- Local:
 - See before +
 - Swelling of the thenar eminence.
 - Edema of the dorsum of the hand (marked).

Complications

- See before + Spread to deep mid palmar space & adjacent web space.

Treatment

- See before +

Incision

- Drained through an incision at the site of maximal tenderness or where pus points at the skin.
- An incision is done along the medial side of the thenar eminence should stop 2 cm distal to the distal wrist crease to avoid injury of the motor branch of medial nerve.

Key points:

- Anatomy of thenar space.
- How to incise.

- may be incised at web space.

spaces of the hand. ← سوال

Superficial Mid Palmar Space Infection

Anatomy

- Anterior → palmar aponeurosis.
- Posterior → flexor tendons.

Etiology

Organism

See before

Predisposing factors & Route

Clinical Picture

Symptoms

See before

Signs

General

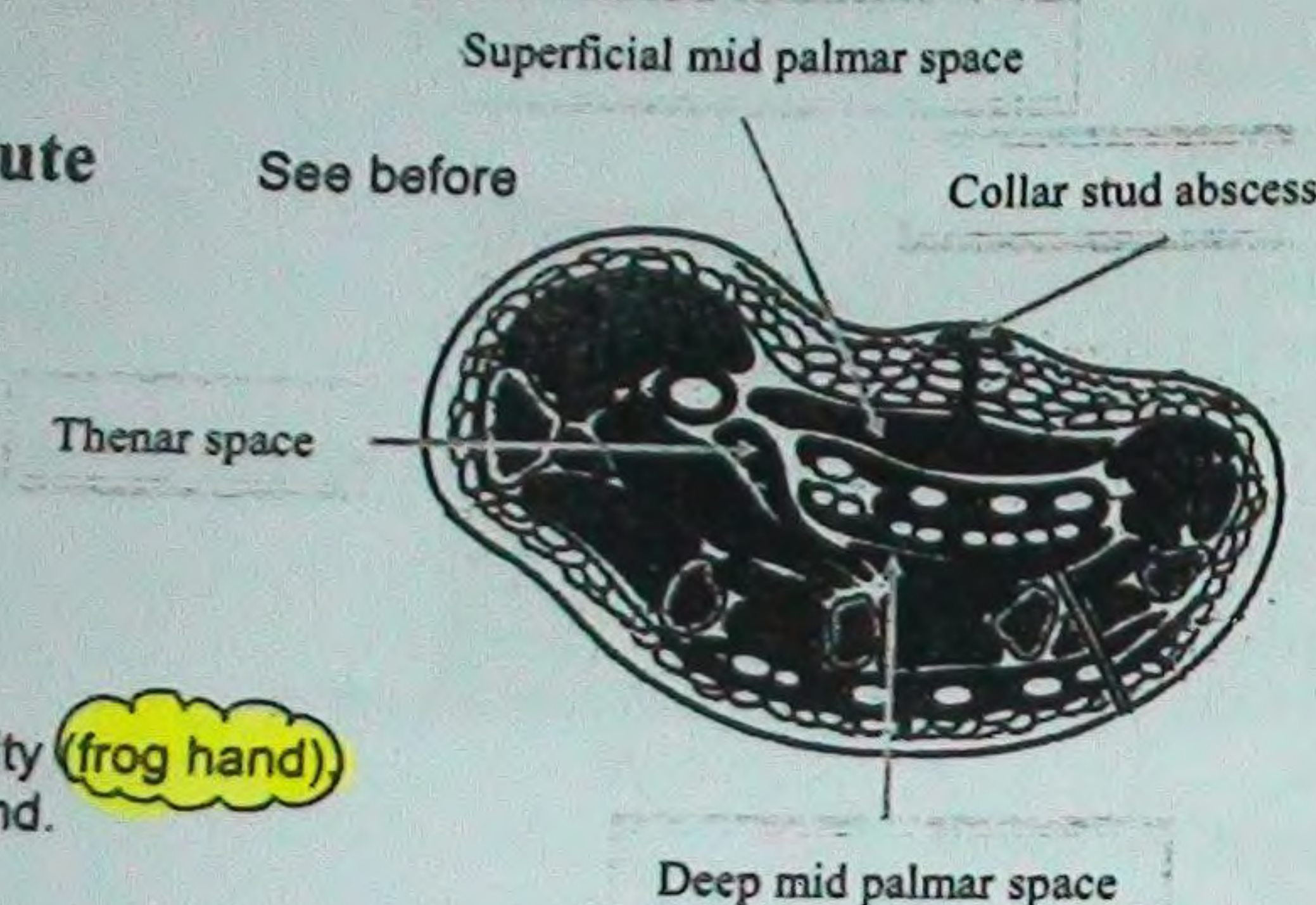
- See before.

Local

- See before +
- Obliteration of the palm concavity (frog hand).
- Edema of the dorsum of the hand.

Complications

- See before +
- Formation of Collar stud abscess:
 - > Locule lies in the SC tissue → subcuticular whitlow.
 - > Locule in the superficial mid palmar space.
 - > Hole in the palmar aponeurosis connecting them.



Treatment

Incision

- A transverse incision at the line of the crease passing over the site of maximum tenderness. → Then examine in proper light
- The palmar fascia is divided in longitudinal direction to avoid injury to digital N. & V. (Hilton's method).

Key points:

1. Anatomy of mid-palmar space.
2. Specific clinical picture.
3. May be complicated by collar stud abscess.
4. How to incise.

hilton
Technique

Then examine in proper light
search for defect if present.
insert a probe widening it then insert artery forceps.

Deep mid palmar Space Infection

Anatomy

- Ant → flexor tendon of the medial 3 fingers.
- Post → fascia covers the interossei.
- Lat → fibrous band from palmar aponeurosis to 3rd metacarpal.
- Medially → fibrous band from palmar aponeurosis to 5th metacarpal.

Etiology

Organism

- See before.

Predisposing factors & Route

- See before.

Clinical Picture

Symptoms

- See before

Signs

- **General** → see before
- **Local** → see before +
 - Obliteration of the concavity of the palm (frog hand).
 - Edema of the dorsum of the hand.

Complications

- See before + - Web space infection of the medial 3 webs - Thenar space infection

Treatment

- see before +

Incision

- Transverse incision over the space
- Counter incision from the web space → if web space is affected
- Semiflexion of the medial 3 fingers

Key points:

1. Anatomy of deep mid-palmar space.
2. Specific clinical picture.
3. How to incise.

Subcuticular Whitlow

- It is pus under the cuticle within the epidermis.
- It may be communicated with deeper abscess (collar stud abscess).

Treatment

- The raised epidermis is incised
- Gentle probing is done for a track extending to a deeper abscess if present.

④ check the palmar Aponeurosis

Collar stud Abscess

Ingrown Toe-Nail (Onychocryptosis)

ضمير الرجل كبر لجوه اللحم
F.O.B
Infection

Definition

Common problem in which a sharp edge of the big toe nail impinges on the adjacent skin fold leading to necrosis of the nail sulcus due to persistent contact of the nail edge.

Incidence

Young males predominate

acute / chronic

Site

It occurs more in the big toe.

Etiology

1. Faulty nail trimming → oblique trimming of the nail sides may leave behind a sharp spike that starts the condition.
2. Wearing tight shoes → thrusts this spike in the soft tissues
3. Nail abnormalities as hypercurved nail.

Complication

This leads to infection & suppuration in the nail fold → may be made worse to cut away the nail.

Treatment

Conservative

(In early cases)

1. A pledget of gauze soaked in an antiseptic is inserted beneath the ingrowing part of the nail to raise it up and to ease away the injured tissues → The pledget is changed daily until the nail grows past the nail fold.
2. Correct trimming of the nail (square nail trimming where the centre of the nail is kept at the same level or even shorter than its edges).
3. Avoid tight shoes.
4. Keep the foot clean & dry.

Operative

Indications:

1. Failure of conservative treatment.
2. Suppurative cases

Technique:

4. Wedge (segmental) excision germinal matrix is the definitive treatment.
5. Excision of the nail with periosteum.
6. If the area is heavily infected, the wound is left open to granulate, otherwise its edges are loosely approximated.

Excision line

Nail portion removed



1ry intention

والضغده هي فضل
لصغر من اخواته

طبيب ولو على الناعيتين بمشايه كنه وبعيش من غير خيف

	Type of incision
Acute paronychia	- Oblique incision at the angle of the nail fold - U - Shaped incision
Pulp space infection	- At point of maximum tenderness - Or anterolateral on the lateral side of the distal 2/3 of distal phalanx - Counter incision → in severe cases.
Web space infection	- A transverse incision over the web one cm from the free margin. - Counter incision posteriorly → severe cases
Thenar space infection	Curved incision done along the first web space
Superficial mid palmar space infection	A transverse incision at the line of the crease
Deep mid palmar space infection	- Transverse incision over the space - Counter incision from the web space → if web space is affected
Tenosynovitis of middle 3 fingers	- Transverse over proximal cul de sac - introduce a ureteric catheter → irrigate with penicillin
Ulnar bursitis	- Along radial border of hypothenar eminence - In severe cases → counter incision at the lower part of forearm
Radial bursitis	- On the ulnar side of thenar eminence - Severe cases → counter incision



Chronic Cat Gut - from intestine of cat. + Chronic
Tensile strength. &
Delays its absorption 10D

Chapter 6 : Surgical infections

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Acute specific surgical infections

1. Tetanus.
2. Gas gangrene.
3. Necrotizing fasciitis

سؤال مهم جداً

1. Tetanus

Definition

- Acute specific infection leading to ↑ nervous excitation & muscle contraction due to release of neurotoxin.

Incidence

- Annual incidence: 0.5-1 million case.
- Predominant disease of undeveloped countries.
- Its incidence is decreasing due to compulsory immunization in childhood.

Etiology

Causative organism

- Clostridium tetani:

- I) Gram +ve, anaerobic bacilli with terminal spore giving drum stick appearance.
- II) It lives in the intestine of horses & the spores are present in the soil & street.

animal
human
faeces

Predisposing factors

- 1- ↑ organism content: (as in contaminated wounds occurring in fields and streets)

- 2- ↓ O₂ content:

- a- From air:

- Deep lacerated wounds.
- Associated pyogenic infections.

- b- From blood:

- Hemorrhage and shock (general cause) → hypoperfusion → anaerobic.
- Lacerated devitalized wounds
- Compartmental syndrome

Local

- c- Lack of proper sterilization of catgut and instruments

دخول ممرضات / متشيلوش / لوشيلت
recoil → providing anaerobic.
Condition for infection

Route of infection

- 1- Wounds: → especially occurred in fields or streets (contamination with animal faeces is common)
- 2- Post-operative tetanus: → lack of proper sterilization of cat gut.
- 3- Tetanus neonatorum: → infected instrument (septic delivery). — incubation periods. → 2-21
- 4- Infected umbilical stump.

Pathology

- ▶ Bacillus at site of inoculation → secrete exotoxin → reaches & spreads through blood &/or motor nerves → reaches CNS.

- ▶ Once it reaches CNS:

- 1- It is fixed to motor cells.
- 2- Can't be detected in blood or CSF.
- 3- Can't be neutralized by antitoxin.

Neurotoxins

Saliva 1.5 L.
Gastric juice 2.5 L.
Bile 800-1200 ml.

Intestinal secretions 1 Day is 8 Liter.

164 *Stenotrophomonas* 2.5 L.

General Surgery

- ▶ **Exotoxin** increases the excitability of motor cells of spinal cord & medulla, so minimal stimuli produces severe spasm.
- ▶ The local effect of the disease is caused by exotoxin effect on **motor nerves**.

N.B.:

- The organism secretes an exotoxin (neurotoxin) which has two actions:
 - 1) **Anti-Choline esterase action**: → Interferes with destruction of acetylcholine at the motor end plate → **tonic rigidity of muscle**.
 - 2) **Extreme excitability of the motor neurons** (ant. horn cells): → Leading to **convulsion attacks** on exposure to minor stimuli.

Clinical picture

Incubation period

- According to previous immunization:
 - Immunized → from 11 days to several weeks or months.
 - Non-immunized → 1 - 15 days.

Symptoms

A. General: FAHM + the patient is alert, vague and mild symptoms as headache, fever, anorexia & rapid pulse.

B. Local:

- Pain → at site of wound.
- Swelling → local small with minimal infection reaction.
- **Convulsions**: the time between appearance of the 1st symptom and occurrence of the 1st reflex spasm is called **period of onset** (if < 48 hours → **bad prognosis**).

Signs

A. General:

1- Stage of tonic contraction (prodromal stage):

- MCQ** → **Trismus** (earliest sign) → spasm of masseter & temporalis muscles.
- **Risus sardonicus** → spasm of facial muscles → **bitter smile** (painful smile)
- **Opisthotonus** → Stiffness of neck & body arched backwards.
- Dysphagia → spasm of pharyngeal muscles.
- **Dyspnea & stridor** → spasm of laryngeal & respiratory muscles.
- Hesitancy in micturition d.t. sphincter spasm may be seen.

2- Stage of clonic contraction: (Reflex convulsion)

- It occurs in more severe cases.
- **Clonic spasm on top of tonic spastic muscles** (i.e. relaxation is incomplete between contractions).
- Excited by light, noise, or any other external stimuli.
- **Muscles are spastic in between attacks** (violent & continuous).
- **Profuse sweating**.

B. Local (wound examination):

- Red, hot, tender & small (± infected with other organisms).

1. **Exhaustion** (from severe convulsions).
2. **Hyper-pyrexia** (excessive energy production) only if convulsions.
3. **Respiratory muscle spasm**.
4. **High cardiac output heart failure**.
5. **Pneumonia**.



Chapter 6 : Surgical infections

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Colonic contraction

1. Strychnine poisoning.

Spasm starts in extremities, complete relaxation between intervals

2. Hydro-phobia in rabies

- History of dog bite.
- Spasm on seeing or drinking water.

1. Tetany

- Carpopedal spasm.
- +ve Chvostek's sign.
- Low calcium level.

2. Trismus

- Excluded by careful local examination.

Tonic contraction

3. Meningitis

- Neck muscles are affected 1st.
- CSF is turbid, contains leucocytes & organisms.

Investigations

- ABGs.
- Organ profile.
- **CBC**: leukocytosis (may be present).
- **CSF**: normal.
- Smears from wound → Gm +ve spore forming organism with **drumstick appearance**.
- **Spatula test** is a bed side test done during stage of tonic contraction:
 - Simple: involve touching oropharynx with spatula (no adverse effects).
 - If normal: gag reflex.
 - If tetanus: reflex spasm of masseter & bite of spatula.

Individuals who previously received 3 or more doses, the last dose was within 10 yrs

Tetanus prone wounds

Only a booster dose of tetanus toxoid (0.5 ml IM)

Individuals who are not previously immunized

Clean minor wounds

1st: 0.5 ml of tetanus toxoid.
Then, 2 further injections are given at 4 weeks intervals

Tetanus prone wounds

1st:
 • 0.5 ml of tetanus toxoid + 250 units IM of tetanus IG (TIG).
 • Antibiotics should be used
Then, Plan to complete the toxoid series

Antitoxins can only neutralize the circulating toxin only not those attached to the nerves.

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General Surgery

Treatment

► Invasive treatment in established cases should be started very soon & respiratory failure may advance very rapidly.

1. Neutralize toxin with TIG. The dose in established cases is 3000-6000 units IM, given preferably in the proximal portion of the wound extremity or in the vicinity of the wound. Repeated doses may be needed.
2. Excise & debride the suspected wound under anaesthesia. the wound must be left open & may be treated with hydrogen peroxide. Surgery should be done after systemic s serotherapy had been started.
3. The patient should be protected from sudden stimuli, unnecessary movements & excitement. Barbiturates are used cautiously as they often cause cardio-respiratory failure. Curarization (muscle relaxation) with mechanical ventilation is a better alternative to large cardiosuppressant doses of barbiturates.
4. The patients with respiratory problems may require tracheostomy. The patient should be intubated once respiratory problems appear.
5. Aqueous penicillin G, 10-40 million units a day.
6. The patient is isolated in a dark quiet room & nutrition is maintained by nasogastric tube.

- Repeated doses may be needed as the half life of antibody is about 3 weeks & tetanus may last longer.
- Diazepam may reduce dose of barbiturates.
- Tracheostomy is done because mechanical ventilation must be continued for weeks.
- Penicillin is given by intermittent IV bolus injection to kill the organisms & prevent further release of toxins.
- All doses should be modified in tetanus neonatorum.

Prognosis

- Death rate is 30-60% in established tetanus with respiratory insufficiency.
- Death rate is inversely proportionate to the length of the incubation period and directly proportionate to the severity of symptoms.
- One attack of tetanus does not confer a life-long immunity.
- Patients who recover from an attack need to have an active immunization schedule.

Tetanus prone wounds:

1. Compound fractures.
2. Deep penetrating wounds.
3. Wounds complicated by pyogenic infection.
4. Wound containing foreign body.
5. Wounds obviously contaminated with soil or dust.
6. Wounds with extensive tissue damage.

- Equine antitoxin should be used only if TIG is not available & only after testing for hypersensitivity.
- Tetanus IG should be taken at a different site & a different syringe from tetanus toxoid.

Vaccination:

- Every child should receive vaccination by DPT (Diphtheria, Pertussis, Tetanus) 2, 4, 6 m & booster at 18 m, by 5 yrs we give only DT → As Pertussis may cause meningitis
- High risk group e.g. house workers, must be vaccinated every 5 yrs.

5 years full immunization → 5 years

5 - 10 years → Booster dose "4DT"

>10 years → not immunized → Antibiotics
① Anti-tetanic
② Start immunization

تفريع الشارب ما يتلفش على

إذا ما أسبغ فينك بربو أو ما تلتا

anaerobic organism

Severe

local & systemic

Response

Chapter 6 : Surgical Infections

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2. Gas Gangrene (Clostridial myositis)

Written

سؤال طبي
عشق

Definition

- Acute specific infection leading to spreading of gangrene with excess gas formation & severe toxemia.

Etiology

Causative organism

- Gram +ve spore forming anaerobic bacilli (that secretes lethal exotoxin).
- Two groups of organisms.

Saccharolytic group (act on CHO)

- Cl. Welchii (Perfringens).
- Cl. Oedematiens.

Proteolytic group: (act on protein)

- Cl. Sporogens.
- Cl. Tertium.

Gas producing organisms

- Etiology:** Cl. species, ↑org., ↓O₂ content
- C/P:** as tetanus + crepitus (gas) + gangrene
- Comp.:** severe toxemia, MOF
- Invest.:** as tetanus + imaging
- TTT:** prophylactic & curative

Bacteriology

5 Marks

Predisposing factors (Closely associated with grossly contaminated war injury)

- ↑ organism content: (as in contaminated wounds occurring in fields and streets)
- ↓ O₂ content:
 - From air:**
 - Deep lacerated wounds specially involving bulky muscles as in gluteal area & thigh.
 - Associated pyogenic infections. → Consuming oxygen.
 - From blood:**
 - Hemorrhage and shock (general cause)
 - Lacerated devitalized wounds
 - Compartmental syndrome
- Lack of proper sterilization of catgut and instruments.**
- Wounds:** → especially occurred in fields or streets (contamination with animal feces is common)
- Endogenous infection:** may occur as in lower limb amputation or large bowel operation or septic abortion.

Crush injury

Pathology

(Anaerobic infections usually do not spread by blood)

A. Local:

- Due to proliferation of the organism & toxin diffuse in surrounding tissue destroying microcirculation → more proliferation.
- Saccharolytic group** → act on glycogen of dead muscles → gases elevates sarcolemma → open the way for invasion with proteolytic group
- Proteolytic group** → act on proteins of dead muscles → NH₃ + H₂S → offensive odor, FeS → stain muscles black

B. General: (Due to exotoxin = alphatoxin)

- Hemolysis of RBCs → slight jaundice.
- Thrombosis of blood vessels → muscle necrosis.
- Severe toxemia and MOF e.g. liver and kidney failure.

that delivers
RBC supply to
the Hs

local

affects all
systems

[except CNS]

Even the immune
system

Severe toxemia
normal
& Leukopenia

containing
amino acid
methionine
Cysteine

جاري

لغزات (Fe)

هنا و هنا

crepitus

Toxin

lethargic

Destroys

phospholipids of cell membrane

hemolysis → Jaundice

Clinical picture**Incubation period**

- Few hours - Few days

Symptoms**A. General**

- FAHM (temperature may be raised).

B. Local

- Patient complains of pain & numbness in affected area.
- Swelling → wound is black & edematous with foul odor with watery discharge.
- Disturbance of function → paralysis of affected organ.

Pain and swelling at the site of a recent wound ± foul-odor discharge

Signs**A. General:**

- Tachycardia: earliest sign (marked).
- Icteric Jaundice → may appear from absorbed toxins, anemia from hemolysis of RBCs.
- MOF (multiple organ failure) → ARF, ARDS
- Shock: dry tongue, hypotension ...etc.

B. Local: (Signs of gangrene)

- The wound shows:
 - swelling, gas bubbles & crepitus sanguinous watery discharge with characteristic foul odour → Spoiled egg
- The affected muscle shows necrosis:
 - Discoloration: red → green → black
 - No contraction if pinched
 - No bleeding if cut
- The surrounding skin may show:
 - Discoloration green or black
 - Blebs full of foul smelling dark fluid.

infectious

Round Sweet

**Complications (Causes of death)**

- Severe toxemia and multi-organ failure.

DD

1. Infection with non-clostridial gas forming organism (usually mixture of Gram -ve bacilli (E.coli) & Gram +ve cocci)

- Not as virulent as gas gangrene.
- Responds well to incisional drainage.

2. Surgical emphysema: (Gas under the skin).

- No signs of toxemia.
- No signs of peripheral circulatory failure.

- The wound looks healthy.

Investigations

- **TLC:** Leucopenia & anemia.
- Raised serum bilirubin (hemolysis).
- Smear & culture from the discharge → gram +ve bacilli.
- Plain X-ray will show gases in the tissue planes.
- CT - MRI: for better evaluation of tissue planes.

Treatment

Prophylactic (All clostridial infections are preventable).

A. Management of the wound:

- a. Skin is incised
- b. Deep fascia is opened
- c. Muscle debridement
- d. Wash the wound with H₂O₂
- e. If less than 8 hrs → close the skin loosely without deep fascia.

Delayed 1ry Sutures.

Best Treatment

antitoxic serum
antigangrene serum
دي الاتق
الاهية

Chapter 6 : Surgical Infections

- f. Avoid deep bandage
- g. If more than 8 hrs → the wound is left opened.

B. General

- Sterile instruments & sutures.
- In nosocomial infection → hospital is closed & sterilized.

C. Specific

- Polyvalent anti-gas gangrene serum: 10 CC IM/d for 3 days (doubtful value).
- Antibiotics e.g. penicillin G.

Curative

Management of the wound is the most important initially in treatment

A. General

- R&M → see before +
- Isolate the patient in a separate place.
- Blood transfusion & IV fluids.
- **Hyperbaric oxygen** (at 3 atmospheric pressure for 1-2 h & repeated every 6-12 h) it inhibit bacterial invasion & toxin production but doesn't eliminate the focus of infection (controversial).

B. Specific

- Anti-gas gangrene Ig: 100 CC IV/d for 10 days (of doubtful value).
- Crystalline penicillin: 20 - 40 million U/day.

C. Others

- Massive type → high amputation above all affected muscles
- Localized type → excise the muscles affected + dusting the wound by penicillin powder + free drainage of the wound.

Prognosis

- **Mortality:** 20% it varies according to extent and severity if injury and timing of treatment
- **Limb salvage:** the rate of saving a functioning limbs not favorable, the myonecrosis occurring render some cases inevitable (life-saving amputation).

N.B.: Proper diagnosis of clostridial gas gangrene depends mainly on the clinical picture of the wound & presence of Gram +ve bacilli

3. Necrotizing Fasciitis**Definition**

- Diffuse non-suppurative spreading inflammation & infection extending to deep fascia.

Etiology [mainly immune compromised]

1. Following surgical procedures, e.g. drainage of peri-anal or ischiorectal abscess.
2. Following I.M injection or I.V infusion.

Causative organism

- The most common organism is group A streptococcus
- **Synergism** (staph + strept + E.coli + anaerobes e.g. peptostreptococci...).
- **Associations:**
 - **Fournier's gangrene** of scrotum which may be followed by **Meleny's synergism** hospital infection. "Abdominal wall"

Etiology: mixed (mainly strept.)
C/P: FAHM, pain, swelling, crepitus, discharge, gangrene, MOF
Invest.: CT/MRI, x-ray, lab. tests
TTT: AAA + debridement + organ support

Diabetic Foot

سؤال نظري

لنسيه
مستكة
وحن فير
عراسه

القائد → Subcutaneous fascia.

Mixed Infection
with sluggish immune Reaction.

Predisposing factors

- Debilitating diseases, e.g. DM.
- Immunosuppressive drugs (usually affects immunocompromised patient)
- Malignant neoplasm.
- Shock.

Pathology

- Infection spreads along fascial planes & results in infectious thrombosis of blood vessels passing between the skin & deep circulation → superficial skin necrosis.
- Hemorrhagic bulla is the 1st sign of skin death.
- Fascial & subcutaneous fat necrosis involves an area wider than the skin →

Clinical Picture

- ➔ The patient is alert & fully conscious.

Symptoms

- General: FAHM. Fever (Hib) → being immune Compromised.
- Local: pain & swelling at the site. very

Signs

- General: marked toxemia → fever & tachycardia.
- Local:

- Edema beyond the area of erythema.
- Crepitus.
- Fever (often absent).
- Greysih drainage (dish water pus).
- Pink / orange skin staining.
- Focal skin gangrene.
- Finally → shock, coagulopathy & MOF.

Very Toxic But not severe inflammatory reaction.

- Skin blistering.

حالة واقعة

The most important signs are:

- Tissue necrosis
- Offensive discharge
- Severe pain
- Gas production (when it is caused by a gas producing organism)

DD

- Cellulitis.
- Toxic shock syndrome.
- Gas gangrene (DD by swab from the wound).

Investigations

Laboratory: CBC, electrolytes, glucose, BUN, ABG & urine analysis.

Radiological:

- X-ray (demonstrate gas in tissue plains),
- CT & MRI to determine the presence of necrosis & surgical debridement.

Prevention

- Adequate debridement of the wounds.
- Blood loss replacement to avoid ischemia.
- Antibiotics for contaminated wounds are essential to prevent postoperative necrotizing fasciitis.

Treatment

1. Surgical debridement (removal of necrotic skin & fascia under anesthesia) → followed by skin flap or graft. → لا ينفع الجرح
2. Antibiotic combination (Penicillin with Gentamycin or Amikin®).
3. Blood transfusion, nutritional support & control of DM if present.

St fascial planes HCO

Early necrosis Late suppuration

HCO 83 undermined edged ulcer

يوقت منه غير ما يستحق

being immune Compromised

very

Very Toxic

But not severe inflammatory reaction.

حالة واقعة

Tetanus

- ① **Definition:** → Not imp **Anaerobic infection**
- Specific anaerobic infection
 - Mediated by **neurotoxin**
 - Lead to → Nervous irritability
Tetanic muscular Contractions

② Aetiology **Stop M**

- 1 **Organism:** **Clostridium tetani** gram +ve
Anaerobic Bacilli terminal (anaerobic bacilli with terminal spore)
Spore (drumstick appearance)

- 2 **Source of Organism** **S**
- Organism naturally present in intestine of Horses
 - Spores present in manured soil & dirt
 - Spores resist heating, dryness, Boiling for 5 minutes

3 **Mode of Infection** **M**

- 1) Wound: contaminated by soil
- 2) Umbilical stump: infected catgut, contaminated dressing and powder.

4 **Toxins** **T** Neurotoxin

- 5 **Predisposing Factors** **P**
- 1) Wounds contaminated by horse excreta
 - 2) Presence of FB or associated pyog. infection
 - 3) Wound is low oxygen tension e.g. deeply seated & lacerated wound
 - 4) Wound low Blood supply
e.g. anemia, shock,
Tight bandage
Plaster of Paris

③ Pathology

- 1- neurotoxin is an **exotoxin** produced locally & reach CNS via blood or motor nerves or both.
- 2 Once reach CNS (fixed by motor cells) & then can't be detected in blood or CNS.

Gas Gangrene

- ① **Definition:** **acute spreading gangrene.**
Associated is gas formation & profound toxemia by anaerobic spore bearing Bacilli
anaerobic spore bearing bacilli

② Aetiology → الحساسية يجب أن تكون

- 1 **Organism:** (fall into two groups)
- 1- **Saccharolytic**
Cl. welchii (breaks collagen releasing CO_2) cl. Septicum
 - 2- **Proteolytic**
cl. histolyticum cl. sporagers.

2 **Source of Organism**

- Organism: normal inhabitant intestine of man & animal
Spores: manured land e.g. Field battle- farms

3 **Mode of Infection**

- 1- Wound: contamination of extensive wound in:
- War injuries must be in bulky ms.
- terrorist- attack on civilized person

4 **Toxins & Toxin:** **alpha market** (alpha toxin) Others include: hyaluronidase, lipase hemolysin

- 5 **Predisposing Factor** **B fiat** ④
- 1- lacerated wound involve **Bulky ms** e.g. gluteus B
 - 2- presence of FB or dead tissue F
 - 3- **ischemia** of muscle due to tight bandage, cast, suture undertension injury main vessel I
 - 4- infection is **aerobic bacteria** → (consuming O_2) A
make field suitable for clostridia (anaerobic)
 - 5- Elderly person is above knee amputation & **fecal incontinence** F

③ Pathology: either local or systemic

- ① **Local:** 1- **saccharolytic org cause:-**
Necrosis of ms due to thrombosis of blood vessels and haemolysis of blood.
Ferment glycogen of dead ms: liberation of H_2 , CO_2
liberated BI pigment stains ms Brick red colour

نقطة الضخ
الكثير

- 3- antitoxin can neutralize neurotoxin just before it's localized in CNS.
- 4- lead to excitability of medulla & spinal cord from which mild stimulus cause violent spasm
- 5- Death from toxemia, exhaustion, resp. obst.



2- Proteolytic org. cause:
 Ferment ptn. of dead ms → liberation of H_2S
 $+Fe \rightarrow Fe_2S_3 \rightarrow$ Greenish black discoloration

② Systemic

- Blood hemolysis lead to pallor, Tinge of jaundice
- Degenerative changes in liver, kidney, suprarenal

④ Clinical Picture

① IP: varies from few hours to few days.

② Divided: general, local

1) General: temp. ↑ pulse
 pt is apprehensive (irritable)
 shock, oliguria

2) Local:-

gas: pain, numbness, wound is swollen, crepitus

Gangrene:

- serosanguineous discharge.
- muscle: Brick red

Don't contract on pinching

If cut not bleed

Greenish black

- overlying skin: greenish black
- offensive odour

الجلد أخضر مسود

④ Clinical Picture

① IP: non immunized pt 24h- 15 days

Imm -- 11 day may weeks or months

② divided into stages:

① Toxemia: ↑ temp. ↑ pulse

Pt. Being irritable, headache, rigor

Pt- gets Hepatitis, myocarditis, gn

(shifts between tonic and clonic)

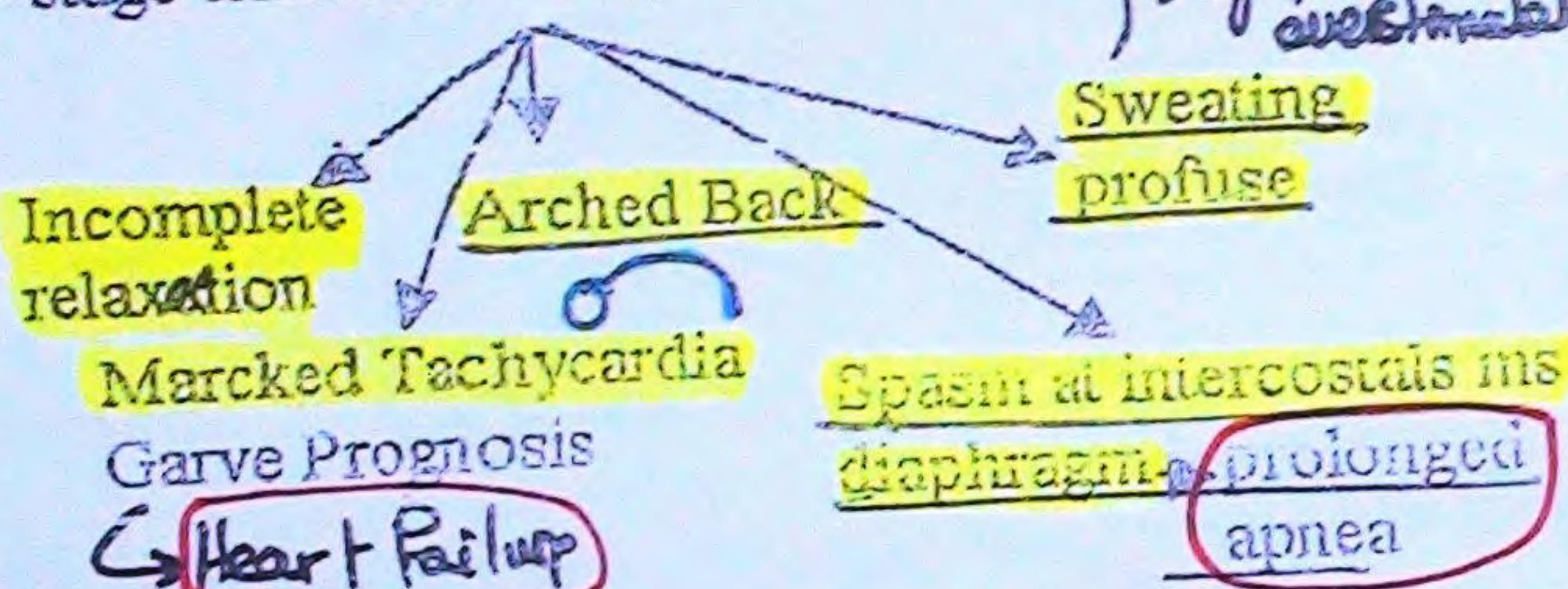
② Tonic:

Pain, numbness, lock jaw, neck stiffness, bitter smile

risus sardonius

③ Clonic:

- violent ms. Contraction reflex to minor stimulus e.g light
- stage characterized by



⑤ Investigation

Polymorphnuclear leucocytosis is present

⑥ Investigation

Mainly, diagnosis based on clinical appearance at wound.

DD. it msr

1) trismus: due to local cause → e.g T.M.J arthritis

2) Tetany carpo-pedalspasm

3) Meningitis: neck muscle 1st

4) Strychnine poisoning: complete relaxation

5) Rabies → history of a dog bite
 spasm on seeing or drinking water

Mainly MS. Of Deglutition & resp.

⑥ DD: A } other cl. Infection

1) simple contamination not significant infection

2) gas abscess:

- noninvasive, no ms. involvement
- itt: incision, drainage

3) cl. cellulitis

- SC, no ms involvement
- edema, gas, skin discoloration

4) Localized cl. myositis:

- non invasive
- myositis

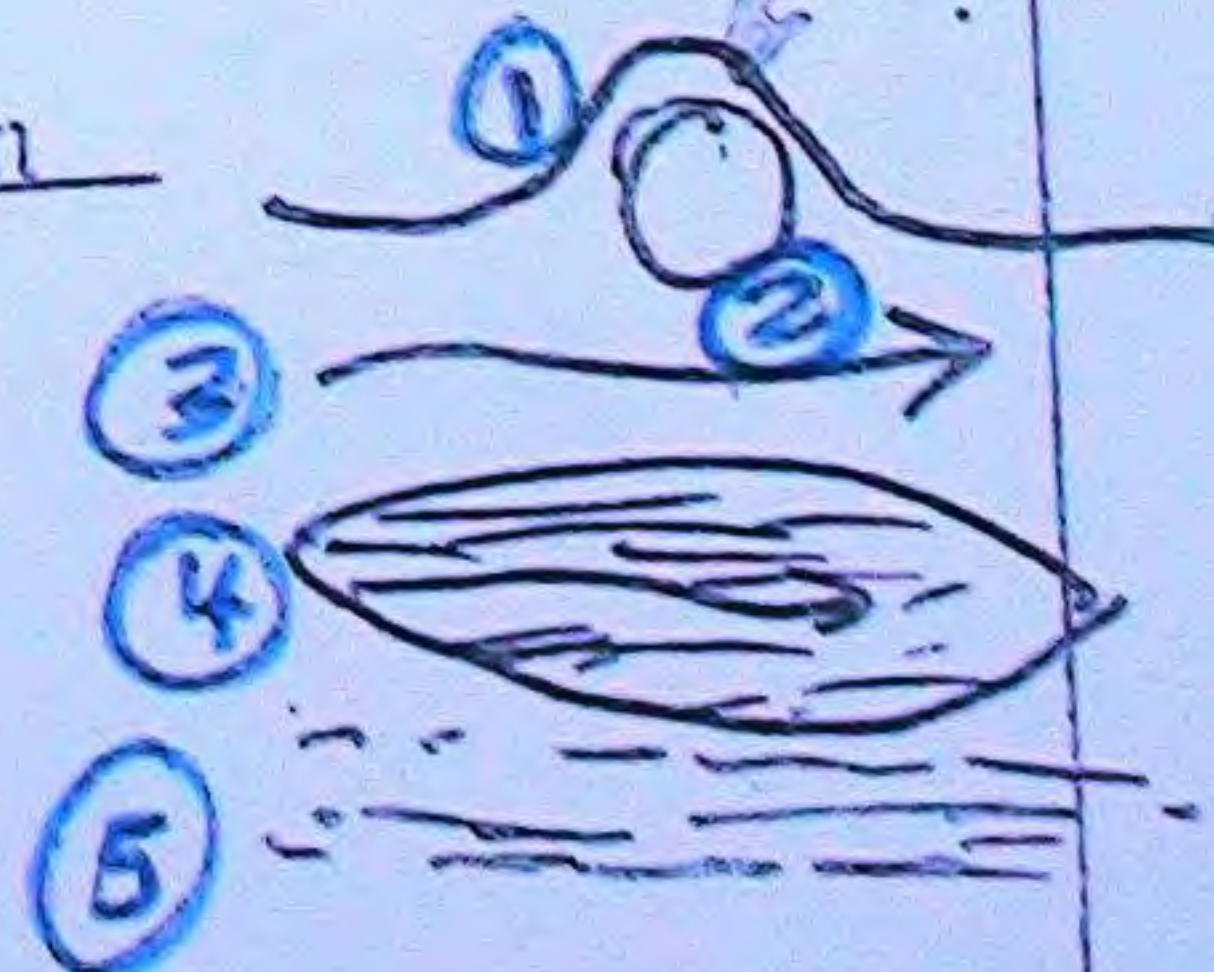
5) oedematous gangrene:

- Highly fatal dies in toxemia
- Cl. oedematiens
- (No gas)

B) Non cl infection (gas forming)

Mixture of gram -ve bacilli & gram +ve cocci

C) c- Surgical emphysema: presence of gas under skin



⑦ Complication

Due to toxemia

- myocarditis

- GN

- Hepatitis

- Avulsion fracture in bone

⑧ Clinical subtypes

See notes

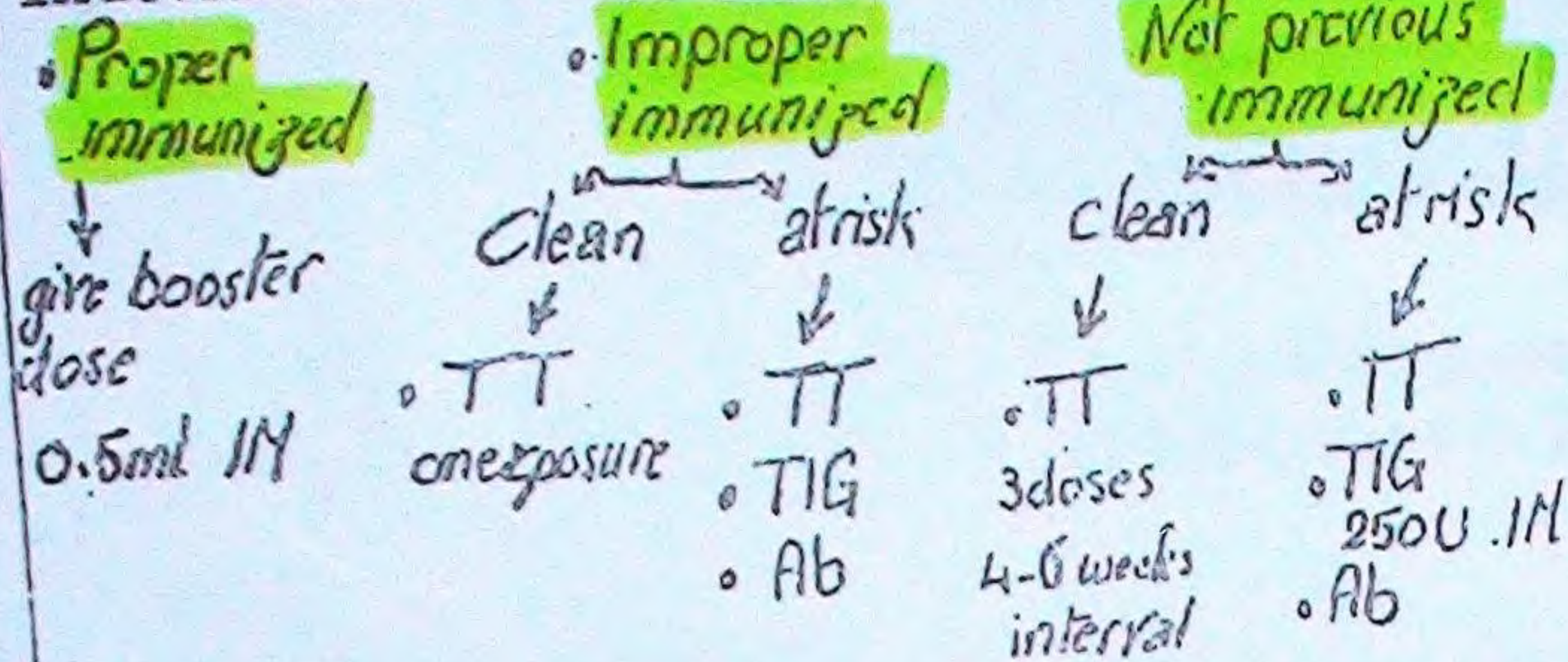
AH page 28

study from book.

Prevention

Every child should be actively immunized by tetanus toxoid and continuing booster injection every 7-10 years

Individual on wound exposure



Prevention

All clostridia infection are preventable.

- adequate debridement of wound, clean, left open
- antibiotics: penicillin
- adequate circ. support avoid tissue hypoxia
- (Anti GG serum isn't used ممنوع)

Treatment:

Intensive ttt should be started soon

1- neutralize by antitoxin TIG 3,000- 6,000 unit IM give in proximal portion of wound or in vicinity of wound, repeated doses may be required

2- Excise & debride:

after neutralize of Toxin, wound left open & H₂O₂ may needed → keep cord. aerobic.

3- the pt.

- Aqueous penicillin 20- 40 million unit/ day
- (Barbiturates) should be cautiously used to avoid CRS failure (depression)
- Curarization & mechanical ventilation.
- Don't disturb pt by unnecessary movement & excitement.
- Dark quite room

N.B: One attack of tetanus doesn't give life long immunity

Treatment

1) Wound Management (Under GA)

- 1 - Dead Tissues & muscle are excised
- 2 - Decompression of Tight fascial compartment
- 3 - deep fascia, skin left open
- 4 - daily exam & debridement is necessary
- 5 - diverting colostomy in extensive perineal infections
- 6 - diffuse myositis & complete loss of blood supply or when a dequate debridement leave useless → amputation

2) Hyper baric oxygen →

bacteria invasion & toxin production given for 1-2 hrs, repeated every 6-12 hrs.

3) Fresh blood transfusion (early given)

4) Antibiotics: penicillin 20-40 million unit/day clindamycin metronidazole can be used

Prognosis

Mortality rate 20% (Many of the 80% left will be amputated.)

Many thanks to Dr/ Aly Hassib

Mohamed Farouk "Rico"

Abd El Rahman Youness



Before



After

SYSTEM

نصيحة!
متحيز

Fungal Infections

Candidiasis

Causative organism

- Candida albicans & other candida species.

Predisposing factors

- Immune suppression (e.g. AIDS & D.M.)
- Broad spectrum antibiotics specially for long duration.
- Hospitalize surgical patients.

Clinical presentation

- Any of these:
 - Oral lesions (Red spots on mucus membranes → become covered with whitish membrane).
 - Candida of the esophagus → dysphagia.
 - Candida of the digestive system → diarrhea & other digestive symptoms.

Treatment

- Oral non-absorbable antifungal e.g. nystatin for oral candidiasis.
- In severe infections → Systemic antifungal therapy, e.g. amphotericin B, 5-fluorocytosine & fluconazole

Antibiotics in Surgical Infections

Introduction

- They are drugs that kill microorganisms but not normal cells.
- They are two groups:

Antimicrobials → Synthetic → As INH, sulpha, quinolones.

Antibiotics → Natural →

- They are secreted from organisms to kill other organisms.
- They are either bacteriostatic or bactericidal.

Choice of antibiotic depends on

Patient:

- Allergy to antibiotic
- Renal & hepatic function.
- Resistance of the host
- Age
- In female: pregnancy, lactation & other drugs e.g. oral contraceptive pills.

Causative organisms:

According to C&S

- Bacteriostatic -

Stop proliferation
Give the immune system
chance to eradicate
infection.

- Bactericidal
Kill the bacteria

لوا ديت الالاتين مع بعض (ماتت بغير مش مع بعض)
بيعاندا بعض

antagonism.

$$1 + 1 < 1$$

Guidelines before prescribing antibiotics

- Antibiotic should be effective against the most likely pathogen.
- Good diagnosis: samples must be taken & C&S is done.
- The change of antibiotic to another is based on C&S & response of patient.
- Length of antibiotic course is based on pathology, clinical & laboratory assessment.

Common antibiotics in practice

1. β -lactam antibiotics:

- Mode of action: inhibit synthesis of bacterial cell wall → lysis.

Classes:

a. Penicillins:

- Common agents: Penicillin G, Penicillin V, Ampicillin, Amoxicillin and Methicillin.
- Spectrum: Gm+ve (mainly) e.g. staph & streptococci & Gm-ve bacteria.
- Ampicillin lacks activity against some staphylococci but active against enterococci & some Gm-ve bacteria.
- Methicillin lacks activity against some staphylococci (Methicillin-resistant staph. aureus, MRSA)

b. Cephalosporins:

- 1st generation Cephalosporins e.g. cephalothin → active against Gm+ve bacteria but not MRSA.
- 2nd generation Cephalosporins e.g. cefamandole → active against Gm-ve but less active against Gm+ve.
- 3rd generation Cephalosporins: very effective against Gm-ve but less against Gm+ve infection.
- Some agents in 2nd and 3rd generations are effective against anaerobes.

c. Monobactams:

- e.g. Aztreonam → active only against Gm-ve aerobic bacteria.

d. Carbapenems:

- e.g. Imipenem → active against Gm+ve, most of Gm-ve and anaerobes.

2. Aminoglycosides:

- Mode of action: interferes with bacterial protein synthesis.
- Common agents: Gentamycin, Tobramycin & Amikacin.
- Spectrum: Gm-ve aerobic organisms (mainly) with little activity against Gm+ve and anaerobic organisms.
- Side effects: ototoxicity & nephrotoxicity.
- Precaution: monitoring of serum creatinine & drug level.

3. Quinolones:

- Common agents: Nalidixic Acid, Norfloxacin & Ciprofloxacin (fluroquinolone).
- Spectrum: Gm-ve aerobic bacteria (most commonly used in UTI).
- Side effects: chondrolytic & nephrotoxic, teratogenic.
- Precaution: not used before 18 years old, in pregnancy and lactation.

4. Glycopeptides:

- Mode of action: interferes with bacterial cell wall synthesis.
- Common agents: Vancomycin.
- Spectrum: Gm+ve bacteria even MRSA & anaerobic bacteria (the drug of choice for treatment of antibiotic-induced colitis which is caused by over growth of clostridium difficile).

5. Tetracycline & Chloramphenicol:

- More active against **anaerobic organisms**.
- Their use in surgical patients has been replaced by more effective drugs.

6. Co-trimoxazole:

- Effective against a variety of Gm-ve aerobic bacteria & some unusual infections in the immunosuppressed patients as those caused by pneumocystis carinii & nocardia asteroides.

7. Metronidazole:

- Excellent **anaerobic activity** but no effect against aerobic organisms.

8. Erythromycin (Macrolides):

- Active mainly against **Gm +ve bacteria** & is usually used in patients sensitive to β -lactam agents.

9. Clindamycin:

- Very effective against **anaerobes** & has some **Gm +ve activity**.

Complications

1. Hypersensitivity reactions especially with Penicillin and Streptomycin.
2. Vitamin B deficiency due to alteration of bowel flora.
3. Superinfection e.g. with proteus and candida.
4. Specific toxicity e.g. bone marrow toxicity with Chloramphenicol.
5. Emerge of resistant strains with mal-use of antibiotics e.g. MRSA.

Antibiotics combination

• additive $1+1 = 2$ • Synergism = $1+1 = 3$

Examples:

1. Bacteriostatic + Bactericidal $\rightarrow$ poor effect.
2. Bacteriostatic + Bacteriostatic $\rightarrow$ Bacteriostatic effect. Except with trimethoprim, sulphamethoxazole $\rightarrow$ Bactericidal.
3. Bactericidal + Bactericidal $\rightarrow$ bactericidal.

The combination may be:

- a. Agonistic (additive or synergistic effect)
- b. Antagonistic.

Indications for combination:**Mixed infections as:**

- Peritonitis due to perforated PU (aerobic, anaerobic).
- After hysterectomy (aerobic, anaerobic).
- We can use Metronidazole + Ampicillin + Garamycin.

To get synergistic effect of the drugs as:

- Ampicillin + Garamycin. *البسيتين به كد (garamycin)*
- Trimethoprim + Sulphamethoxazole.

To overcome bacterial tolerance

- To overcome resistance of staph. $\rightarrow$ Amoxicillin + Clavulanic acid (Augmentin®) or Ampicillin + Sulbactam (Unasyn®).

To prevent development of resistance

- Treatment of TB $\rightarrow$ by INH + Ethambutol + Rifampicin.

Penicillin $\rightarrow$ cell wall.
+
aminoglycoside $\rightarrow$ DNA

Decontamination

See operative book.

Introduction for any Burn Qs

Burns

معدل جرحاً جرحاً جرحاً جرحاً
• Classification, • Complication, • Management

- Burns is one of the most serious injuries to the body.
- They cause dramatic damage to the physiological functions of the skin, which lead to serious local or systemic complications.
- By size and weight, the skin is the second largest organ in the body next only to the muscles.
- Loss of large amounts of skin. If not replaced, is incompatible with life.

الرجل واليد
شخصية وحيدة

Etiology:

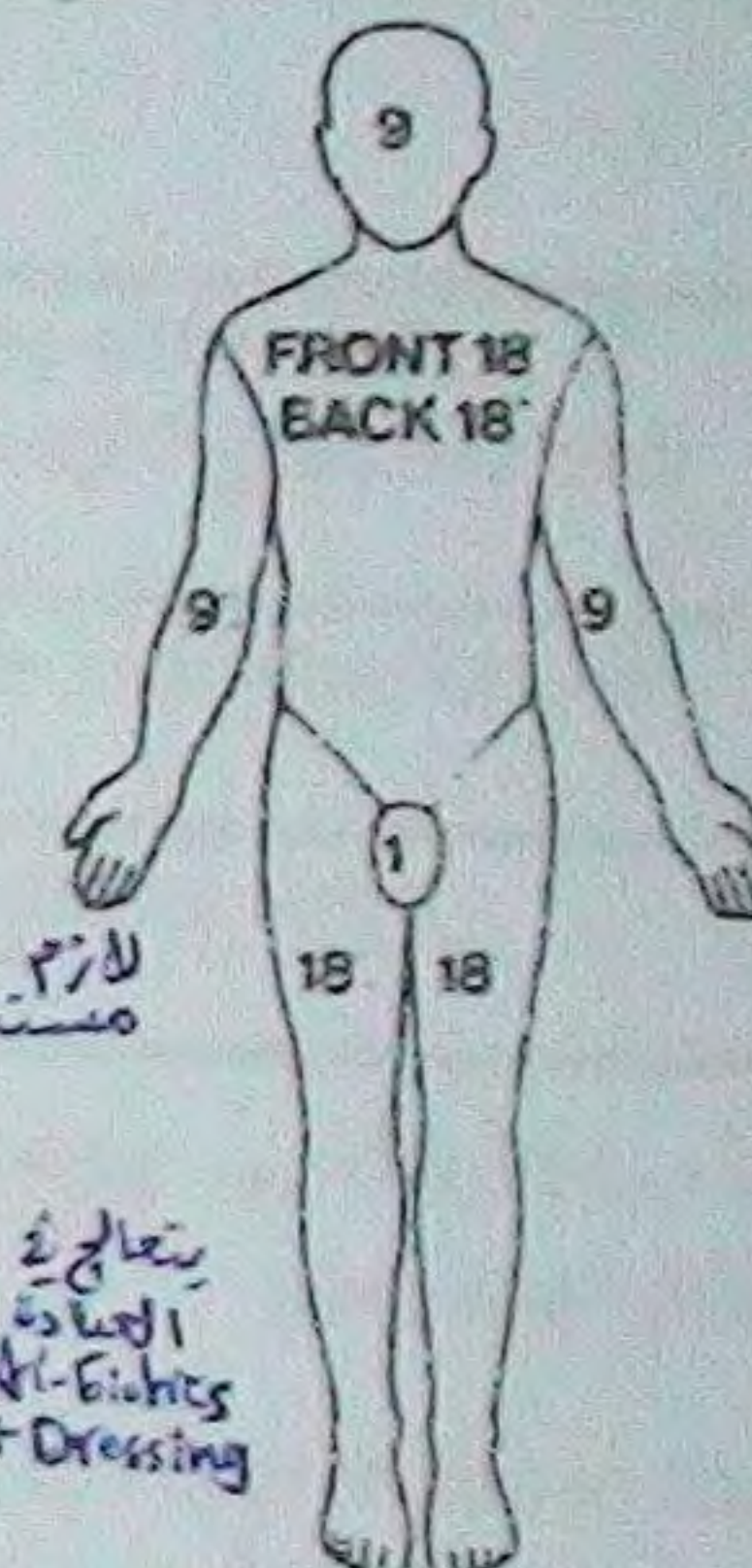
1	Scalds	Caused by boiled liquids.
2	Flame burns	Often due to ignition of clothing.
3	Electric burns	Blood & bones are good conductors, thus every organ in the body may be affected.
4	Chemical burns	Are due to either acids or alkalis.
5	Inhalation burns	Due to exposure to hot gases and may affect the upper or lower airway.

Classification:

[A] Classification according to the percentage surface area involved:

- This follows the rule of 9.
- In children the rule of 9 needs some modification due to the large size of the head in comparison to the rest of the body.
- Burns are classified into:

1	Major burn	Involves more than 30% of the body surface area.
2	Intermediate burn	Involves 15-30% in adults & between 10-30% in children.
3	Minor burn	Less than 15% in adults & 10% in children.



لازم
مستشفى
يتعالج في
العيادة
Anti-Biotics
+ Dressing

[B] Classification according to the depth of the burnt tissues:

1. **First degree burns:** The usual example is sunburns. Only the epidermis is damaged causing erythema of the skin. They heal rapidly.

2. **Second degree burns:** The epidermis & a portion of the dermis are damaged. If no infection occurs, epithelial regeneration can occur from the remnants of hair follicles and sweat glands in the dermis and the burn will heal in 3 weeks. If infection occurs, these epithelial elements are destroyed and the case will be changed to a full thickness burn.

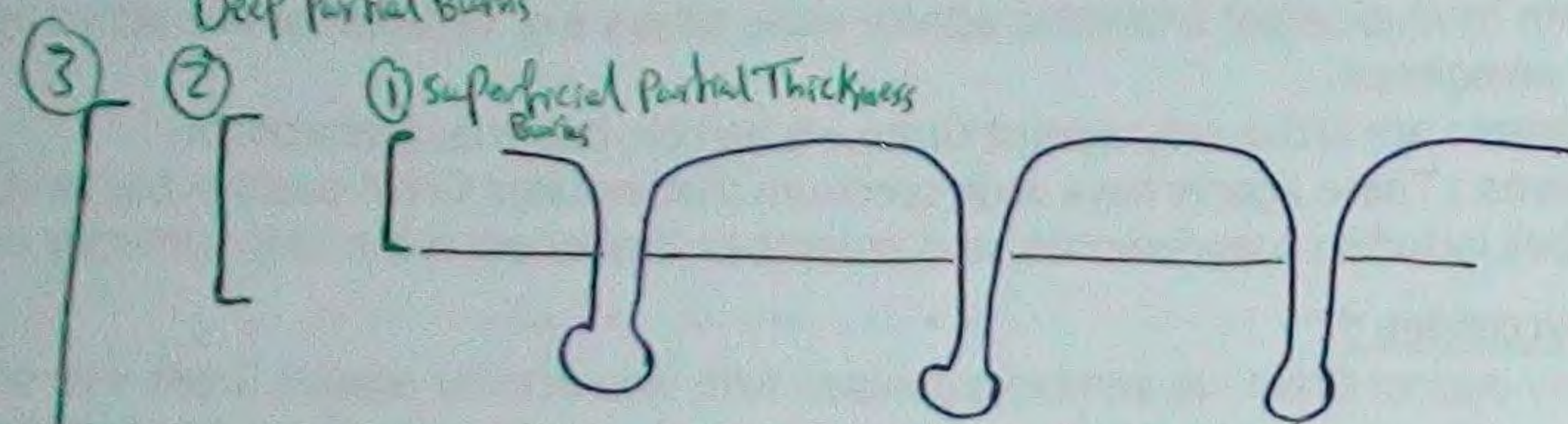
- This degree is further subdivided into:
 - (a) **Superficial partial thickness burns:** Only the epidermis is damaged causing erythema of the skin. They heal rapidly.
 - (b) **Deep partial thickness burns:** The epidermis and a portion of the dermis are damaged.

3. **Full thickness burn:** There is complete destruction of the epidermis and dermis. After separation of the eschar by the third week, the patient should be prepared for skin grafting.

- The differentiation of partial thickness from full thickness burn is sometimes difficult and even in the same area.

لا يمكن
الشفاء
الشفاء
الشفاء

Full Thickness Burns
Deep Partial Burns



① → Re-epithelialization بسرعة

② → =

③ → =

3 weeks

Epidermis
ما
الغالبية كالماتة
لا يمكن
الشفاء
الشفاء
الشفاء

ذلك ولا ده
Appendages
لأنه عايشين
لأنه عايشين

في كثير من الأحيان التفرقة بين (Partial thickness) و (Full Thickness) يكون صعباً ← وينتشر نسيجه لغاية (Healing) ونسوف حرق كل ما يلي و (Hair follicle) ولا في حرقه في (ulcers)

لحرق الحرق ← تكون قشرة اسف (Eschar)

General Surgery

AH

- Both patterns may intermingle together. However, the following may help to differentiate:

Partial thickness burns	Full thickness burns
1. Mottled red.	1. White or black eschar. (ميتة)
2. Moist due to exudation of plasma. + sweat gland	2. Dry.
3. Shows blisters surrounded by erythema. فقاعة	3. Possible visible thrombosed subcutaneous vessels.
4. Very painful and sensitive to air (pin-prick test).	4. Painless due to loss of the terminal nerve endings.
5. Should heal within 3 weeks.	5. Granulation tissue formation and eschar separation (starts to occur after 3 weeks).

Epilation test: Useful test is to pull on a hair. If the hair pulls easily & painlessly, the burn is a deep one.

Histopathology:

- Temperatures greater than 45°C results in protein denaturation, which exceeds the capacity of cellular repair.
- Thermal injury to the skin results in 3 distinct zones:

1	The central inner zone (zone of coagulation)	If left, the eschar separates within 3 weeks leaving either a bed of granulation tissue (in full thickness burns) or a re-epithelialized bed (in partial thickness burns).
2	The intermediate zone (zone of stasis)	May die over the next 48 hours post-burn, if tissue oxygenation not maintained.
3	The outer zone (zone of hyperemia)	Tissues in this zone normally recover within 7-10 days unless subjected to infection.

- The rate of healing of burns depends on the surviving hair follicles and sweat glands. لأن صنادول الأولاد إلى بيطلعوا جلد جديد

Pathophysiology:

- Increased capillary permeability leads to the loss of enormous amounts of fluids & proteins in the damaged area. (It is maximum in the first 8 hours) and continues for 48 hours. * أول يومين
- Excessive loss of water by evaporation with loss of calories.
- The burnt area will be colonized by bacteria.

* Complications of burns:

1- Systemic complications:

1. Inhalation injury:

- Asphyxia may occur immediately from inhalation of heat or gases.
- Atelectasis, pneumonia, emphysema and pulmonary edema may follow.
- Finally respiratory failure may occur with systemic sepsis.

2. Neurogenic shock:

is caused by pain.

3. Hypovolemic shock:

may develop if the fluid losses are not properly corrected.

4. Renal failure:

Acute renal failure can occur after prolonged uncorrected hypovolemia.

5. Gastro-intestinal complications:

(a) Acute ulceration of stomach and duodenum (Curling's ulcer) are rarely seen nowadays with the routine prophylactic use of antacids.

(b) Ileus usually occurs during the early post-burn period.

(c) Acute ulcerations of the colon.

6. Multiple organ failure

often follows sepsis. The outcome is poor.

Burns in Upper 1/2 is More Dangerous than Lower 1/2

أنا بأخاف من infection في واحد محروقة

Partial Thickness

Full Thickness

* احنا لازم نعرف المحروقة ده

غالبًا لو جملته بكماله في اليوم الرابع

+

غالبًا لو جمل infection ← يبقى لازم آجل ال graft

فأعمل له ال graft قبل اليوم الرابع (3-5 days)

← جيتا بعد أول يومين ما كنت بحلقه Re-suscitation في أول يومين

لأن عيانه Major Burn ← غالبًا بيكون Shocked

* Criteria for Admission to Hospital,

1- Burns in Hands, Feet, Genitalia

2- Circumferential Burns

3- Restricted Ventilation

4- Burns to Joints

5- Electrical Burns

6- Associated organs Disease "HF, Kidney F., Liver F."

7-

General Surgery

AH

II- Local complications :

[A] Early local complications :

1. **Infection** : Is the primary cause of death in burnt patients. Infection may lead to the development of septicemia and septic shock. It occurs usually between 4-7 days post burn. Treatment is essentially by prevention through proper local burn wound care and systemic antibiotic therapy.
2. **Constricting eschars** : May form in deep circumferential burns of the limbs and chest and should be treated by urgent escharotomy (release).
3. **Edema** : In burns of the face and neck, edema may lead to suffocation and urgent tracheostomy may be needed.

• معظم الوفيات سببها Septicemia

[B] Delayed complications :

1. **Contractures across joints** : Splintage and immobilization in the "best functioning position" with physiotherapy is important to prevent contracted scars.
2. **Scar formation (hypertrophic or keloid)**.
3. **Malignant transformation (Marjolin' ulcer)** : In long standing unstable scars.

Management of burns :

[A] First aid management :

1. A patent airway should be assured.
2. A strong analgesic as 50mg pethidine is administered I.V.
3. Tetanus prophylaxis.
4. Saline or tap water, at room temperature, can be poured over the burnt area for 15 minutes to decrease edema and relieve the pain.

[B] Minor burns : (Less than 15% in adults and 10% in children)

- Can be treated as outpatient & treatment consists of :
1. Dressing, using the proper local chemotherapeutic.
 2. Analgesia and systemic antibiotic prophylaxis.

[C] All majors and most moderate burns (except very superficial ones) should be admitted to the burn unit. :

1. A wide bore I.V. cannula is inserted rapidly.
2. A Foley's urethral catheter is introduced to check the urine output.
3. Treatment essentially consists of :

I- Resuscitative fluid therapy :

The amount and rate of fluid replacement :

- Depends on the weight of the patient and the percentage of the total body surface area injured.
- It is essentially give for the first 48 hours and after that.

The amount : The amount infused during the first 24 hours is 2ml/percent surface area burn/kg body weight. Half the amount calculated is given over the first 8 hours and the other half over the next 16 hours. Also half the calculated is given over the second day.

The types : The types of fluids infused vary in each formula.

1. Evan's Formula :

- 1st day : 1 ml/kg/% burn normal saline + 1ml/kg/% burn colloid (Dextran) + 2000 ml glucose (Provided the daily caloric needs).
- 2nd day : 1/2 ml/kg/% burn normal saline solution + 0.5 ml/kg% burn colloid + 2000 ml glucose.

2. Parkland's formula :

- 4ml/kg% burn lactated Ringer's solution/day.
- 1/2 the amount is given in the first 8 hours, 1/4 in the second 8 hours, and 1/4 in the third 8 hours.

It is to be noted that :

- In all formulae, the maximum percentage of burn calculated is 50%; otherwise serious over infusion may occur.
- Administration of blood can be started after 48 hours guided by the hematocrit value.
- Oral intake is avoided during the first 48 hours to avoid gastro intestinal complications and is started gradually after that.

How to judge the adequacy of intravenous resuscitation ?

1. Regular check-up of vital signs.
2. The urine output should be 30-60 ml/hour.
3. C.V.P. in critical cases.

N.B.: Patients who have extensive burns are liable to have a serious catabolic status. Introduction of intravenous hyperalimentation has made it easy to correct this problem and to support the patient nutritionally during this critical period.

II- Local burn wound care : (After resuscitation)

- **Constricting eschars** (in the limbs and chest) have to be released immediately. **Urgent fasciotomy** in deeper burns may be limb saving.
- **The aim** of the local wound care is to avoid infection.
- After cleansing & conservative debridement, **topical antimicrobial agents** should be applied.
- **The wound is managed by either :**

1. The exposure method :

- Exposure method requires isolation of the patient in (completely aseptic atmosphere)

Advantages :

1. It is more comfortable to the patient.
2. It avoids the need for repeated change of dressings, which implies a great burden on nursing staff and is rather expensive.
3. It inhibits the growth of bacteria by the dry air surrounding the burn.

Indicated for :

1. Burns of the face, neck and perineum.
2. Burns involving one side of the trunk.

2. Bulky occlusive dressing (the occlusive method) :

Dressing change in the occlusive method can be done by the use of a special tub (Hubbard Tank) or under anesthesia (especially in children) since it is usually very painful and adherent.

- Both occlusive and exposure methods are equally effective.
- All partial thickness burns should heal within 2-3 weeks.
- Full thickness burns will require closure by autogenous skin grafts.

[D] Later care :

Following the previous regimen of wound care, all partial thickness burns should heal within 2-3 weeks, while full thickness burns will require closure by autogenous skin grafts. For deep burns :

1. Autologous skin grafting :

- Partial thickness skin grafts (Thiersch grafts) are commonly used to cover the large raw areas produced by burns.
- A graft is applied after either spontaneous separation of eschar (3-4 weeks), or early burn wound excision.
- **Early burn wound excision** is the preferred method for deep burns of the hand.

• Patients are usually subjected to surgery 3-5 days post-burn (before bacterial infection sets in), where tangential excision of the damaged dermis is carried on. When healthy bleeding tissues are reached a skin graft is applied.

2. Biological dressings :

• Indications :

1. Autograft is not enough.
2. Local wound condition is not favorable.

• Advantages :

1. The wound will be less painful.
2. Minimize fluid and protein loss.
3. Control infection.

• Examples :

1. Allografts (Cadaver's skin).
2. Xenograft skin (Pig's skin).
3. Amniotic membrane.

- Applied after removal of the eschar and are changed every 3-4 days.
- It is only temporary and permanent closure can be achieved only by autograft skin.
- Artificial skin substitutes are used with promising results.

3. Prevention of contractures : Contraction is a normal component of wound healing and its prevention across joints can be achieved by proper splintage, immobilization in the best functioning position, and physiotherapy.

Prognosis of burns

[A] Burn factors :

1. Extent : This is the most important factor. Mortality is about 50% if the extent of burn is 50%.

2. Depth : Deeper burns carry higher mortality and more disfigurement.

3. Site : Burns of the face are the worst because they are likely to be accompanied by inhalation burns of upper and lower respiratory tracts.

4. Infection : Septicaemia is the major killer of burn victims.

5. Type : High-voltage electric burns are the worst. They are usually fatal. (Scalds with boiling water commonly cause superficial burns and are the least harmful.)

6. Associated injuries.

[B] Patient factors :

1. Age : Burn victims at extremes of age (children and elderly) have bad prognosis.

2. Concomitant diseases e.g. diabetes and coronary heart disease.

[C] Treatment factors :

Patients who are treated in **specialized centres** have better chance of survival and avoidance of complications.

Special Types of Burns

[A] Electric burns : (A form of thermal injury)

- **Severity of the burn :** It is divided into high and low-tension injuries according to the voltage responsible for the injury (whether above or below 1000 volts).
- **Tissue damage is due to :** the passage of the electric current through blood and bones.
- **The affected blood vessels** usually show late thrombosis with delayed progressive ischemic necrosis at different sites.

[B] Chemical burns :

- **Tissue damage is due to :** Oxidation, reduction, corrosion or protoplasmic poisoning.
- **Severity of the burn :** is determined by the strength of the agent, its amount, duration of skin contact and its mechanism of action.
- **Management :**
 1. Systemic control by administering the proper antidote in case the agent is absorbed.
 2. Locally, the agent should be thoroughly cleansed; all saturated clothes are removed and affected areas irrigated with massive amounts of water.

Treatment of special burns

1. Burns of the face : The aim :

- (a) Is to keep the skin movable.
- (b) To care for the eyes and ears.
- (c) Obtain good cosmetic result :

* Upper Airway

it's Managed by Exposure Method

- Where the burn extends into the scalp, the hair is shaved for at least 2 inches beyond the burn. The burn is best treated by exposure but deep burns require early grafting either after initial excision or as soon as the eschar begins to separate.
- In burns of the eyelids, the splinting action of the crust may lead to exposure keratitis, which should be avoided by the frequent application of Vaseline or cold cream. If the eye is injured, the conjunctival sac should be irrigated with normal saline and cocaine hydrochloride is instilled to alleviate the pain.
- In burns of the ears, infection should be avoided to prevent exposure of the cartilage.

2. Burns of the hand :

- The usual treatment is by occlusive dressing with the application of a plaster slab to ensure immobilization and facilitate elevation.
- The dressing is left for 12 - 15 days by which time superficial burns are healed but deep burns will require grafting after a further 10 days of preparation.

* Keep Hand in the Functioning Position

3. Burns of joint surfaces :

- Exposure treatment with hydrotherapy and physiotherapy is the best method.
- In deep burns, early skin grafting is essential to obtain a good functional result and avoid contractures and deformities.

Managed by occlusive dressing

4. Burns of the perineum :

- The best treatment is by exposure and topical silver sulfadiazine applications.
- In burns of the penis, the external meatus should be attended to, in order to prevent stricture, and in burns of the vulva a pledget of gauze or cotton wool should be inserted between the labia to prevent adhesions.

Management of Burns

A & T

Management of Burns

Prepared & Typed by: Ahmed ElSherbiny
Ahmed Bravo

1st aid management:

A₂T₃

- 1- Airway: should be maintained patent.
- 2- Analgesic: should be strong as 50ml pethidine IV.
- 3- Tetanus prophylaxis.
- 4- Tape water or saline at room temp. poured on burnt area for 15 min. to decrease edema and relief pain.

Minor Burns:

- 1- **Criteria:** less than 15% in adults
less than 10% in children
- 2- Treated as out patient.
- 3- A Analgesics
- 4- A Antibiotics (systemic)
- 5- Dressing using proper local chemotherapeutic.

Major Burns: & most moderate (except superficial)

- 1- **Criteria:** Major: more than 30%
Moderate: Adults 15% - 30% & Children 10% - 30%
- 2- Administered to burn unit.
- 3- C Canula: wide bore I.V. canula is inserted rapidly
- 4- C Catheter: folley's urethral catheter is inserted to monitor urine output.

Resuscitative Fluid Therapy

Amount and rate of fluid replacement depend on the weight of the patient and % of total body surface area injured.

- Amount infused during the 1st 24 hours is 2ml/percent surface area burn/kg bodyweight. Half the amount calculated is given over the 1st 8 hours & the other half over the next 16 hours. Also half the amount calculated is given over the 2nd day.

Formulas:

Evan's Formula: 1st day: 1 ml/kg/% normal saline + 1 ml/kg/% colloid + 2000 ml .. glucose. (the daily caloric needs)

2nd day: 1/2 ml/kg/% normal saline + 0.5ml/kg/% colloid + 2000 ml glucose

Modified Brook's Formula:

1st day: 2-3 ml/kg/% lactated Ringer + 2000 ml glucose

2nd day: 1 ml/kg/% lactated Ringer + 0.5 ml/kg/% colloid + 2000 ml glucose

Parkland's Formula:

1st day: 4ml/kg/% lactated Ringer: 1/2 amount over 1st 8 hours
1/4 amount over 2nd 8 hours
1/4 amount over 3rd 8 hours

Duration: It is essentially given for the first 48 hours.

Monitored by:

- 1- Regular check up of vital signs
- 2- Urine output should be 30-60 ml/hour
- 3- C.V.P

N.B.:

- Max. % calculated in all formulas is 50% to avoid serious over infusion.
- Blood administration can be started after 48 hours guided with hematocrit value
- Oral intake is avoided in 1st 48 hrs to avoid GIT complications.
- Extensive burned patients are liable to have serious catabolic status. I.V. hyperalimentation makes it easy to correct this problem and to support the patient nutritionally during this critical period

Local Wound Care

- 1- The aim is to avoid infection.
- 2- Constricting eschars 2be released immediately.
- 3- Urgent fasciotomy in deeper burns may be limb saving.
- 4- Topical antimicrobials should be applied after cleaning & conservative debridement.

Wound is managed by one of the following:

1. **Exposure method:** requires isolation in aseptic atmosphere.

Advantages: a. more comfortable to patient.
b. avoid repeated change of dressings.
c. inhibits bacterial growth by dry air.

Indications: a. Burns of face, neck & perineum.
b. Burns involving one side of the trunk.

2. **Bulky Occlusive dressing (the occlusive method):**

Since it is usually very painful; dressing change can be done by Hubbard Tank or under anesthesia (especially in children).

- Both methods are equally effective
- All partial thickness burns should heal within 2-3 weeks
- Full thickness burns require closure by autogenous skin grafts.

N.B. Biological Dressings: (N.B)

Indications:

- a. Autograft is not enough
- b. Local wound condition is not favorable.

Examples:

- a. Allografts (cadaveric)
- b. Xenograft Skin (Pig's)
- c. Amniotic membrane.

Advantages: a. Wound will be less painful.

b. Minimize fluid and protein loss. c. Control infection.

- Applied after removal of eschar and are changed every 3-4 days.
- Only temporary and permanent closure only by autograft skin.
- Artificial skin substitutes are used with promising results.

Surgery

Time: 3-5 days post-burn (b4 bacterial contamination)

Method: tangential excision of damaged dermis & grafting is done either immediate or late when healthy granulations appear.

Indications: Deep burns

Prognosis Of Burn

Burn related factor:

- a. Extent: rule of 9 (discuss)
- b. Depth (discuss)
- c. Type of burn: high tension electric burn has the highest mortality rate
- d. Site of burn (upper half is worse)
- e. Infection (discuss)
- f. Associated injuries

Patient Related:

- a. Age: extremes have bad prognosis.
- b. Coexistent cardiopulmonary diseases.

TTT Related Factor:

ttt in special burn centers gives better prognosis.

when should I wait for late grafting?

- 1) infection
- 2) doubtful diagnosis (partial or full)
- 3) if the burned area is wider than the capacity of grafting

A patient should change the dressing if it becomes wet → to avoid culture of infection. (too much change of dressing is not good also as it affects the epithelium formation.)

Principles of skin coverage

1- Skin Grafts

Definition

- It is segment of epidermis & dermis that has been completely departed (cut) from its blood supply & donor site → transferred into a recipient site (skin loss)

Types

- Split thickness graft (**Thiersch graft**)
- Full thickness graft (**Wolfe graft**)

Steps

- Separation of the required segment by surgical knife (dermatome)
- The recipient area should be surgically clear & has formed granulation tissue (capable to regenerate due to adequate blood supply)
- After application of the graft:** The process of **(TAKE)** takes place which aims at nutrition of the graft by:
 - Imbibition of plasma (48 hrs) from newly blood vessels
 - Vascularization (after wards)
- Healing of donor site:** depends on the degree of thickness split or full.

- **Types:** split, full thickness
- **Split:** Trunk, Ihigh, ↑Take, ↑Trauma
- **Full thickness:** Full dermis, Face, Foot, For good cosmesis & sensation



	Split thickness	Full thickness
Definition	Epidermis + superficial part of dermis	Epidermis + full thickness of dermis
Indications	1- Covering large area of granulation tissue. 2- Coverage after: a) Deep burn. b) Malignant tumor resection.	1- Facial wounds 2- Palmar aspect of hands and plantar aspect of feet. 3- Site of pressure on sole of the foot
Donor sites	1- Trunk, thighs 2- Upper arm, forearm	1- Post-auricular skin 2- Upper eye lid 3- Supraclavicular
Advantages	1- Early separation & application. 2- Increase TAKE by graft. 3- Can cover wide area. 4- Early detection of recurrence of malignancy. 5- Donor site heals spontaneously.	1- Direct closure of donor site. 2- Minimal contraction. 3- Better sensation. 4- Cosmetically better. 5- Resistant to trauma.

Disadvantages

- | | |
|---|---|
| 1- More liable contraction.
2- More liable for pigmentation.
3- Weak resistance to trauma.
4- Poor sensation.
5- Cosmetically poor. | 1- Limited donor site.
2- Favourable granulation tissue.
3- Less TAKE.
4- Asepsis must be perfect.
5- Scar at the donor site. |
|---|---|

- ✓ Healing in split thickness graft occurs according to the degree of thickness
- Minimal → 1 week
 - Intermediate → 2 weeks
 - Thick → >2 weeks

Whenever you take skin graft

The following points should be put into consideration:

- 1- Healing by 1ry or 2ry intention
- 2- Color of the graft.
- 3- Skin appendages (hair follicle, sweat glands, sebaceous glands)
- 4- Sensation of the graft.
- 5- Durability of the graft.
- 6- Growth of the graft.

Graft survival

- Phase I (24 – 48 hours) → plasmatic imbibition.
- Phase II (2 – 4 days) → insoculation → Recipient bed capillaries align with those of the graft.
- Phase III (5 – 7 days) → Revascularization → anastomosis & neovascularization → it needs immobilization of the graft with mild compression.
- ▶ The thinner the graft, the faster is the take.
- ▶ Bare bone, bare cartilage & bare tendon are all bad recipient sites for grafts (poorly vascularized), a flap is better applied.

2- Flaps

Definition

- Tissues to be transferred from one site of body to another one with their blood supply.

Indications

- Wound with poor vascular bed (e.g. overlying bare bone, bare cartilage, bare tendons)
- Reconstruction of facial features.
- When sensation is required.

Types

I- Skin flaps

- Can be classified into:

A- Local flaps:

▣ Random pattern flaps:

- These flaps have no anatomically recognized blood supply. Thus, they should have strict length to width ratio in order to survive.
- A safe random pattern flap has a length to width ratio of 2:1 maximally.

▣ Axial pattern flaps:

- These flaps are supplied by known arteries, and have no limitations regarding length to width ratio.
- Adjacent nerves can be included, and the flap can be a sensory one.
- Axial flaps may retain their attachment to the donor area and are termed **pedicled flaps**.
- If they retain their blood supply without the skin attachment, they are termed **island flaps**.

B- Distant pedicled flaps:

- ▣ Distant → a flap needed to remote site from donor site e.g. from L.L. to U.L.
- ▣ Pedicled → blood supply reaches through a pedicle which needs to be cut after neovascularization.

- **Indications:** wounds (with ↓vascularity, in the face), sensation is required
- **Class.:** skin flaps (local, distant)
- **advantages:** cosmesis, vascularity, carrier

II- Musculocutaneous (myocutaneous) flaps

- ▣ These are axial flaps that receive their blood supply from the underlying muscles through perforator vessels.
- ▣ The muscle is lifted from its bed with the overlying fascia and area of skin.
- ▣ The incorporation of the pedicled muscle in the flap enhances the survival of the cutaneous component.
- ▣ Famous examples are the latissimus dorsi myocutaneous flap that is used to cover wide defects resulting from wide radical mastectomies, and the other example is pectoralis major myocutaneous flap used to cover defects in the neck or face.

III- Fasciocutaneous flaps

- ▣ They have the same principles as the previous one, but their blood supply is through fasciocutaneous perforators.

IV- Microvascular free flaps

- ▣ It is now possible to anastomose vessels of less than 1 mm in diameter.

Advantages of flaps

- 1- Good color & texture matching
- 2- Provide extravascular supply & sensation
- 3- Allow for future operation on the same area
- 4- can be used on a carrier for bone, muscle, skin

3- Tissue expanders

- Tissue expanders are inflatable silicone implants.
- They are placed subcutaneously in collapsed state.
- Over several weeks the expander is gradually inflated with saline through a subcutaneous port.
- The overlying skin is gradually stretched to accommodate a larger area.
- Finally, the expanded skin can be used to cover defects and the tissue expander is removed.



Aesthetic surgery

Definition

- Surgery to improve appearance for correction of any disfigurement.

Patient selection

- It is very important as there is no pathology to correct, but rather an improvement in the body image is asked for.

Examples

1. Rhinoplasty.
2. Face lifting.
3. Eyelid surgery.
4. Breast surgery (augmentation or reduction mammoplasty).
5. Liposuction (body sculpting surgery):
 - Special cannula with 1 cm incision → connected to high vacuum machine → aspirate excess fat from abdomen, buttocks or trochanteric areas.
 - Autologous fat injection → aspirated fat is reinjected to augment other areas.

Bed sores (Pressure ulcers)

Definition

- A localized area of soft tissue injury resulting from compression between a hard prominence & an external surface. It is a type of avascular necrosis.

Pathology

➡ STAGES:

- I → Non – blanchable erythema.
- II → Partial thickness skin loss "epidermis".
Manifested as: abrasion, blister, shallow crater.
- III → Full thickness skin loss.
SC tissue is exposed (damage or necrosis of SC tissue).
- IV → Muscles or bones are exposed
Tissue necrosis of any supporting structure (muscle – bone – tendon – joint capsule).



Stage I



Stage II



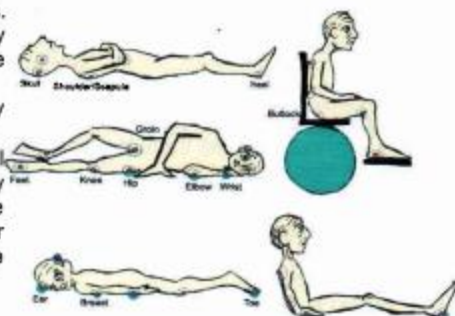
Stage III



Stage IV

➤ SITES:

- 1) Common anatomical pressure points.
- 2) Positional pressure points: by abnormal position of the limbs or the body.
- 3) Instrumental pressure points e.g. by bed rails.
- 4) Internal viscera exposed to unusual pressure e.g. trachea pressed by balloon of endotracheal tube (especially if a ryle is present) or severely distended colon in Ogilvie syndrome.



Etiology

1. Prolonged recumbency.
2. Warmth & moisture of skin (soiling by urine, stool, wet sheets or even generalized or local edema).
3. Shear forces: during manipulation & positioning e.g. rolling of the patient in bed.
4. Bad general condition: e.g. pneumonia, malnutrition or electrolyte imbalance.

Diagnosis

- Frequent inspection of pressure points in high risk patients for early non-blanchable erythema (stage I).

Complications

- a) **General:** Bacteremia, septicemia, toxemia.
- b) **Local:** Osteomyelitis, cellulitis, pyoarthrosis.

Treatment

A. PROPHYLAXIS: (Most important)

1. Reposition every 2 hours.
2. Pressure reducing mattresses.
3. Skin care & improve general condition.

B. DEFINITIVE TREATMENT:

- **Stage I:** as prophylaxis, more frequent turning of the patient.
- **Stage II:** as I + dressing to prevent dryness of the wound for better healing.
- **Stage III & IV:** Debridement:
 - 1) Chemical (small wounds): saline dressing + collagenase (Iruzol).
 - 2) Surgical (large wound): may require myocutaneous flap.

CHAPTER: 7

TRAUMATOLOGY

- 1- Surgical trauma
- 2- Wounds:
 - Wound healing
 - Wounds suturing
 - Wound dressings
- 3- Blood transfusion
- 4- Hemorrhage
- 5- Shock:
 - Hypovolemic shock
 - Septic shock
 - Cardiogenic shock
 - Neurogenic shock
- 6- Infusion therapy
- 7- ARDs

Blast injury.

Major Trauma

"Triage" "Poly-traumatized Patients"

Incidence

- Represent the commonest cause of death among people aged 1-44 years. (youth)
- It is the 3rd commonest cause of death among all age groups.
- Number of permanent disabilities caused by trauma is double the number of deaths.

Etiology

1- Closed (Blunt) trauma:

- Direct → e.g. road traffic accident [RTA] (most common), fall from height & direct blow.
- Indirect: fracture ribs.

2- Open (Penetrating) trauma:

3. Iatrogenic

A. Low velocity:

- Localized over a small area.
- E.g.: knives, sharp objects & bullets.

→ Pistols

B. High velocity:

- (evidenced) (rifles)
- Its energy is decipated over a wide area.
- Shock wave spread-out of the main missile track & affect areas far-away from the 1^{ry} missile.
- The higher the velocity of the missile the more the damage.
- Common example is: fire arm injuries caused by rifles.

Smashed Bones → 2ry pullets

$$Damage = mass \times V^2$$

Pathology & cause of the death

- There are 3 main factors affecting the survival of a polytraumatized patient: Hypothermia, coagulopathy & metabolic acidosis make a vicious circle leading to fatal outcome.

I. Hypothermia:

- Leads to slowing of the clotting factors & coagulopathy.
- Warming the patient by blankets or the use of warm IV fluids is essential.

II. Coagulopathy:

- Leads to diminished tissue perfusion & exaggeration of shock.

III. Metabolic acidosis:

- Due to anaerobic metabolism (disturb enzymatic activity of the cells) → hypoperfusion → anaerobic mechanism → Pyruvic acid.

Causes of death:

- Classified according to timing of death after the accident into:
 - Immediate (minutes): Hemorrhage, associated injuries to vital structures → 50%
 - Early (hours): hemorrhage, major fractures → 30%
 - Late (weeks): sepsis, MOF → 20%

* Organized systems care → prevent early Mortality.

* Critical care → prevent late Trauma Mortality.

urea 20-40 mg%

[BUN] → B. urea Nitrogen

Creatinine 1.2 mg%

• $PO_2 = 95-100$ mm Hg

• $PCO_2 = 35-45$ mm Hg

Rupture Cervical spine → atlas & axis → Death. "Brain stem"
Below that level → Quadriplegia.

- If a moving car stops suddenly, the person will continue moving forward.
- If he is not wearing the seat belt, the head will strike against the car. However, the seat belt itself may cause injury to clavicle, GIT.
- Seat belt injuries: may include fracture clavicle, trauma to the intestine or mesentery. The skin mark should raise suspension.

Classification (TRIAGE SYSTEM) Block → Dead.

- Triage involves sorting patients into three groups by colored labels according to their injury and availability of resources as follow:

1) Red: those who will die anyway whether they receive medical attention or not.

2) Yellow: those who will survive only if they receive timely medical attention. The first hour after injury is called golden hour.

3) Green: those who will survive anyway whether they receive medical attention or not.

- If the number of patients exceeds the resources, the yellow code is treated first → then green.
- If the number of patients doesn't exceed the resources, all codes are treated.

Management of Poly-Traumatized Patient

- The american college of surgeon made ATLAS (advanced trauma life support system) which is internationally accepted protocol.
- It has 3 elements:
 - a. 1ry survey & resuscitation
 - b. 2ry survey.
 - c. Definitive treatment.

A. Primary Survey

Where & when

- Start at site of accident continues as trauma victim reaches the hospital & repeated at the hospital

Objectives

- To identify & treat any immediately life-threatening condition.

Steps

Tube Color

ABCDE

- A. Airway + cervical spine control
- B. Breathing.
- C. Circulation (and hemorrhage control)
- D. Disability. (Brief neurological assessment)
- E. Exposure & Environment.
- Life threatening problems discovered during the 1ry survey (e.g. tension pneumothorax) are always dealt with before proceeding to the 2ry survey.

A-Airway and cervical spine control

- 2 things are done with each others:
 - I. Airway evaluation & protection if needed
 - II. Controlling movement of cervical spine.

I) Airway:

- Assessment: - if the patient is able to speak freely, his airway is patent.
- All the patients receive supplemental O_2 by mask upon arrival.

Cell Death

apoptosis.
shrunken nucleus
exposure of antigenic Determinant
phagocytosed.

necrosis
liberation of Toxins.

HCO Any patient with Glasgow Coma Scale < 8
Below 8 → Intubate

endotracheal Tube

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General surgery

- **Clearing:**
- 1st remove:
 - Vomit, blood or foreign body either manually (finger sweep) or with a rigid sucker.
- 2nd Then do chin-lift or jaw thrust.

Protection: by one of the following methods:

- 1) **Oropharyngeal or nasopharyngeal airway tube:**
 - ▶ Aim: prevents tongue from falling back & occluding the airway.
 - ▶ When: in an unconscious person.
- 2) **Tracheal intubation:** either by:

OROTRACHEAL INTUBATION	NASOTRACHEAL INTUBATION
Allows the usage of large tube	Safer if the cervical spine appears fractured

- ▶ When:
 - ⑤ - Coma Scale < 8
 - Apnoea.
 - Risk of aspiration.
 - Impending or actual airway compromise (inhalation injuries, maxillofacial trauma).
 - Closed head injuries (allow hyperventilation to ↓ ICP)

3) Cricothyrotomy:

- Either by making a cut & inserting a tube or percutaneous insertion of a wide bore needle.
- It is not suitable for children

Tracheostomy is rarely needed in the emergency room management

II) Cervical spine control:

Assessment:

- These categories of patients should be considered with unstable cervical spine & need radiological diagnosis:
 - a. Clinical examination reveals bony abnormalities or cervical tenderness
 - b. Multisystem trauma, a blunt injury above the clavicle or an altered level of consciousness from trauma or from drug / alcohol intake.
 - c. Maxillofacial trauma.

Management:

- a) **Immobilization:**
 - Done using a backboard & a rigid collar
 - If a collar is not available, manual in-line immobilization is necessary.
- b) **Radiological evaluation:**
 - This is done later after stabilization of vital signs, at least 3 views (lateral, AP, odontoid) are done.

B-Breathing

Assessment:

- Assess breathing & search for emergency signs by:
 1. **Inspection:** for air movement, respiratory rate, cyanosis, tracheal shift, jugular venous distension
 2. **Palpation:** for subcutaneous emphysema & flail segments.
 3. **Auscultation:** for upper airway sounds (stridor, wheezing or gurgling).
 4. **Percussion:** for hyperresonance or dullness over either lung field.

Tension Pneumothorax
open Pneumothorax

Cardiac Tamponade. Beck's Triad. → Shock. Muffled heart sound. Congested neck veins.

PEEP → Positive end expiratory pressure ventilator

Chapter 7 : Traumatology

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Management:

- These are life-threatening thoracic conditions that must be diagnosed to save patient's life.

DIAGNOSIS	INITIAL MANAGEMENT	FOLLOWED BY
Tension pneumothorax	Needle decompression	Intercostal tube
Cardiac tamponade	needle pericardiocentesis	Operative pericardiectomy & control of bleeding
Flail chest	Intubation	PEEP
Massive hemothorax	Chest tube insertion	Thoracotomy may be needed if bleeding continuous
Open pneumothorax	occlusive dressing fixed at 3 sites only	chest tube

C-Circulation

Shock:

- 1) Hemorrhagic shock (commonest).
- 2) Cardiogenic shock: cardiac tamponade & myocardial trauma.
- 3) Neurogenic shock: spinal cord injury - pain.

Management:

1. Control bleeding with direct pressure if possible.
2. Two large-calibre (16 gauge) peripheral IV lines are inserted, central line may be needed.
3. Blood samples are sent for typing, cross matching, Hb%, HCT & blood chemistry.
4. Ringer's lactate solution is infused as a start. Volume of crystalloid needed = 3 X estimated blood volume lost.
5. When cross matched blood is available it is given immediately.
 - IV fluids & blood are given at a rate that ensures urine output between 0.5 - 1 ml/kg/hr for adults.

D-Disability

Assessment: (brief neurological assessment)

- Common causes of neurological deficits related to trauma:
 - Head injury.
 - Hypoxia.
 - Shock.
 - Alcohol or drug abuse
- AVPU evaluation:** based on patient's best response
 - A: Alert & interactive.
 - V: Vocal stimuli elicit a response.
 - P: Painful stimuli are necessary to elicit a response.
 - U: Unresponsive.

- Followed by Glasgow coma scale in the secondary survey.

E-Exposure & Environment

- Clothes:** all clothes of the victim are removed using sharp large scissors.
- Warmth:** keeping the emergency room warm & using blankets to prevent hypothermia.
- Insert:**
 - Foley's catheter to monitor urine output (this is contraindicated if there is blood at the urethral meatus).

(suspecting urethral injury)

Missed hemorrhage
intrathoracic
head injury
intracranial

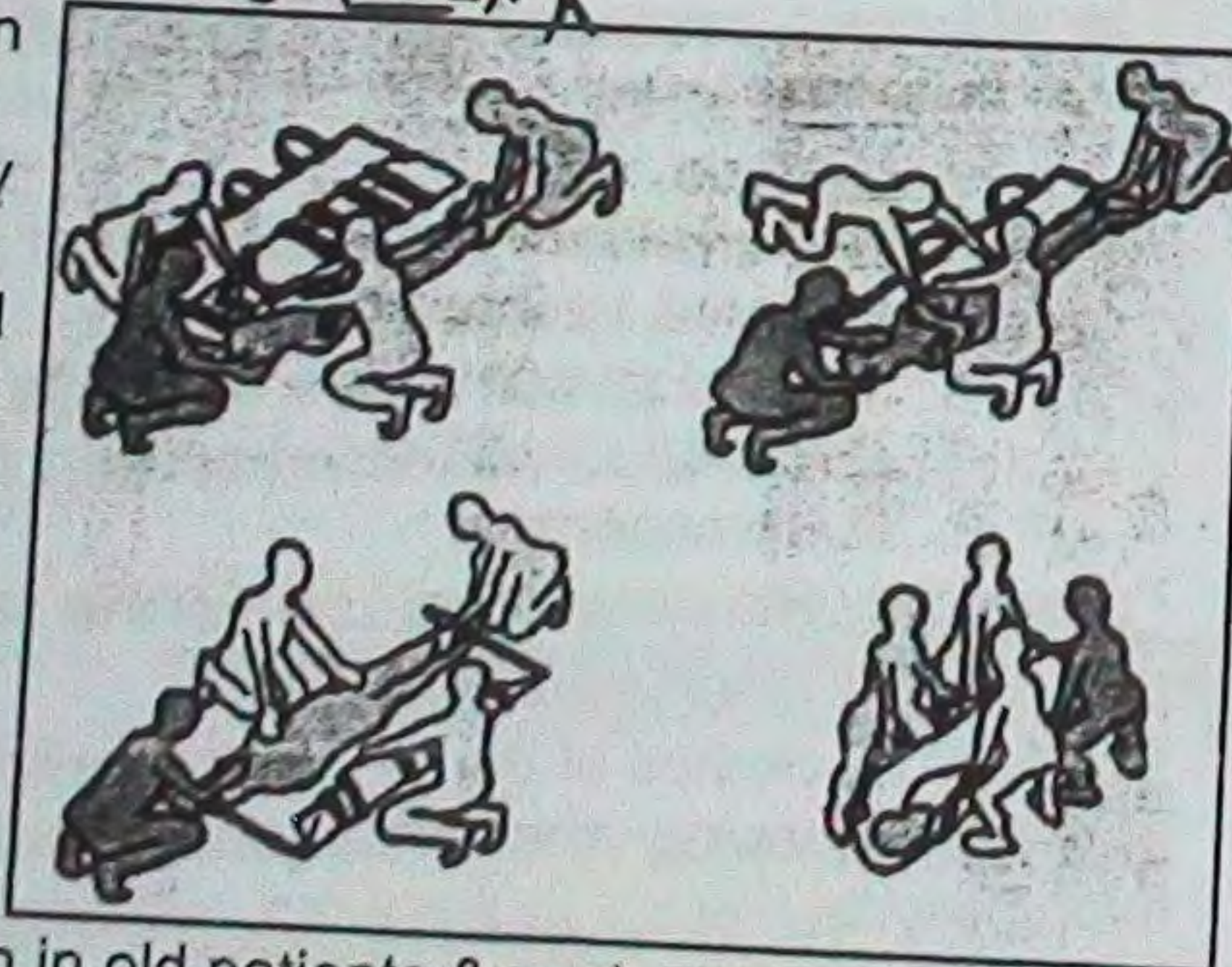
0.5 - 1 mL / kg / h.
30 - 40 mL / h.

General surgery

- Nasogastric tube (Ryle's) decompresses the stomach & prevents vomiting & aspiration. *Preventing acute gastric dilatation. Prevent aspiration.*
- **Radiological assessment:**
 - For blunt trauma → Chest & pelvis X-ray AP view.
 - For penetrating trauma → CXR & X-ray on trauma site.
 - CT scan or X-rays after resuscitation.
- **History: (AMPLE)**
 - ✓ Should be taken while completing 1st survey.
 - ✓ If trauma victim is unconscious, history is obtained from the rescue team, accident witness & family members.
 - ✓ **AMPLE history is:**
 - Allergies.
 - Medications.
 - Past medical history.
 - Last meal (time). *آخر مرة*
 - Events of injury. *ما حدث*

B. Secondary Survey

- Once resuscitation is done & urgent X-rays have been evaluated.
- **Objectives:**
 - Examination of the patient from head to toe & front to back.
 - Taking a complete medical history.
 - Integration of all clinical, laboratory & radiological information.
 - Formulation of a management plan.
- **This phase includes:**
 - 1- Head to toe examination of undressed & stable patient**
 - **Head examination:**
 - Injuries
 - Mouth
 - Eye (pupil → size and reaction)
 - Ear and nose
 - **Neck & spine:** neck collar for fixation:
 - Absent pain or neurological signs does not exclude injury.
 - Move the patient's body as one piece (**log rolling**) by 4 persons.
 - **Chest:** searching for pneumothorax, hemothorax, cardiac tamponade.
 - **Abdomen:**
 - **Indication of Diagnostic Peritoneal Lavage (DPL):**
 - Blunt abdominal trauma in adult, associated with:
 - ✓ a. Suspicion of organ injury with doubtful signs.
 - ✓ b. Unreliable abdominal examination because patient is unconscious.
 - ✓ c. Unexplained hypotension that may be caused by blood loss.



- **Perineum** including rectal examination in old patients & vaginal examination.
- **Neurological:**
 - Glasgow coma scale.
 - Cranial nerve examination.
 - Sensation & motor activity.

Chapter 7 : Traumatology

- Limbs: for fractures and neurovascular bundle.
- 2- **Taking a complete medical history.**
- 3- **Investigations according to needs:**
 - **Laboratory:**
 - HB%, glucose level, Kidney functions, PO₂, PCO₂, Na⁺, K⁺.
 - **Radiological:**
 - Plain X-ray: chest and skeletal or visceral injuries.
 - CT and MRI: chest, abdominal or head traumas.
 - U/S: for abdominal injuries.
 - Duplex for vascular injuries.
 - **Instrumental:**
 - Abdominocentesis or thoracocentesis.
 - GIT or urinary endoscopies.

C. Definitive Treatment

- During secondary survey after stabilization of the patient we can detect the definitive injury by its specific clinical picture and specific investigations and deal with the patient according to the type of injury and priorities.
- During this phase the following should be noted:
 - 1) Some cases may be transferred to another hospital or another department in the hospital.
 - 2) Level of care should not be allowed to drop during transfer.
 - 3) The patient requires repeated evaluation as some injuries may present later (e.g. injuries of spleen & duodenum & subdural hematoma).

Prognosis

- Depends on the severity of the lesion and time passed during arrival to hospital
- Immediate deaths represent 50%, early deaths (1st few hours) represent 30% and only 20% are late deaths due to multiple organ dysfunction syndrome.

Points To Remember

- **At the end of the major survey, detect or exclude the following Five serious problems:**
 1. Airway obstruction
 2. Open or tension pneumothorax
 3. Massive hemothorax
 4. Flail chest
 5. Cardiac tamponade
- **The Five causes for upper airway obstruction:**
 1. Tongue
 2. Blood
 3. Loose teeth or denture
 4. Vomitus
 5. Soft tissue edema
- **When you face a patient with severe bleeding, remember the five areas of potentially severe bleeding**
 1. Chest
 2. Abdomen
 3. Pelvis
 4. Long Bone Fractures
 5. External bleeding
- **Five main features of respiratory distress**
 1. Tachypnea and/or dyspnea
 2. Use of accessory muscles of respiration
 3. Difficult speaking
 4. Low Oxygen saturation
 5. Agitation or confusion
- **Sites of hidden hemorrhage:**
 1. Intra-abdominal bleeding.
 2. Intrathoracic bleeding.
 3. Fractures of pelvis & femur.

DPL: Diagnostic peritoneal Lavage.

Tertiary Survey 3- Re examination to detect any missed injury

CT scan is not used in shock patient.

UCC

① Organ profile.
② Site of Trauma FAUS, focus assisted sonogram in Trauma.
③ in shock patient
④ Complication

Summary of ATLAS protocol

➔ **Iry survey (ABCDE):**

Airway & cervical spine:

- Airway & cervical spine:
- If patient can speak freely then airway is clear, otherwise airway should be cleaned & protected.
 - Cervical spine immobilization & radiological diagnosis is done in some trauma victims.

Breathing:

- Is assessed by clinical examination.
- Manage according to diagnosis.

Circulations:

- If patient is shocked, resuscitation must be done.

Disability:

- Brief neurological evaluation is done using AVPU evaluation.

Exposure & environnement:

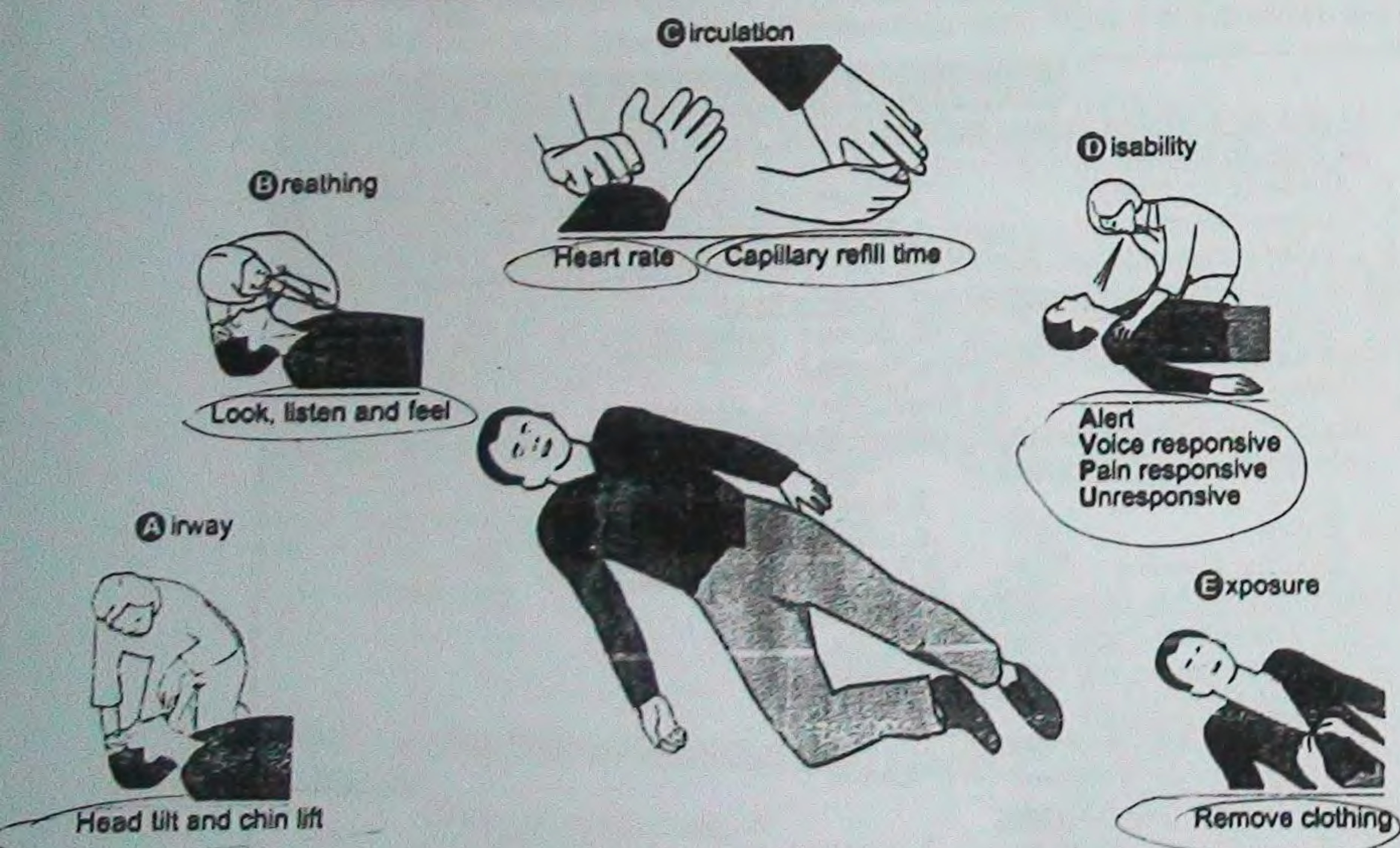
- This includes AMPLE history.

➡ **2ry survey:**

- Head to toe examination.
- Taking complete medical history.
- Integration of all clinical, laboratory & radiological information.
- Formulation of a management plan.

➔ **Definitive treatment:**

- According to type of injury.



Metabolic response to trauma

- The response is characterized by an acute catabolic reaction, which precedes the metabolic process of recovery and repair. This metabolic response to trauma was divided into an ebb and flow phase by **Cuthbertson**.
- The **ebb phase** corresponds to the period of severe shock characterized by depression of enzymatic activity and oxygen consumption. Cardiac output is below normal, core temperature may be subnormal, and a lactic acidosis is present.
- The **flow phase** can be divided into:
 - A **catabolic phase**: with fat and protein mobilization associated with increased urinary nitrogen excretion and weight loss, and $\rightarrow$ -ve nitrogen balance.
 - An **anabolic phase**: with restoration of fat and protein stores, and weight gain.
- In the flow phase, the body is hypermetabolic, cardiac output and oxygen consumption are increased, and there is increased glucose production. Lactic acid may be normal.

Phase	Duration	Role	Physiological	Hormones
Ebb	<24hrs	maintenance of blood volume; catecholamines	- Decrease <u>BMR</u>, <u>Temp</u>, <u>O₂ consumption</u>; vasoconstriction; - Increase <u>Co</u>, incr. <u>HR</u>; acute phase <u>proteins</u>	Catecholamines, Cortisol, Aldosterone
Flow				
Catabolic	3-10 days	maintenance of energy	Increase <u>BMR</u>, <u>Temp.</u>, <u>O₂ consumption</u>, <u>negative nitrogen balance</u>	- incr. glucagon, - insulin, cortisol, catecholamines
Anabolic	10-60 days	replacement of lost tissue	positive nitrogen balance	Growth hormone, IGF

Renal changes

- ❑ Oliguric renal failure.
- ❑ When acute oliguric renal failure complicates trauma:
 - ◆ Retention of → urea, uric acid, and creatinine
 - ◆ Acid products of cell metabolism such as phosphates and sulphates → cannot be excreted.
 - ◆ Liberation of intracellular K⁺ & Mg⁺⁺ → high levels in extracellular fluid.
 - ◆ Metabolic acidosis → manifested by a decreased CO₂ combining power.

ENDOCRINE RESPONSES TO TRAUMA

- ENDOCRINE RESPONSES TO FEVER**
- ☑ The adrenal cortex → aldosterone secretion → ↑ sodium and water reabsorption by the renal tubules.
 - ☑ ↑ Catecholamines secretion → VC, tachypnea & tachycardia.
 - ☑ ⊕ hypothalamus → post. pituitary → ADH secretion → H₂O retention
 - ☑ Ant. pituitary → GH, Prolactin & ACTH secretion
 - ☑ ↑ IL1 (by macrophages) → ↑ body temperature (fever), WBCs & IgG
 - ☑ Thyroxin → ++ sensitivity of Catecholamine.
 - ☑ Glucagon → ++ Glucose.
 - ☑ Renin → Ag I → Ag II
- Handwritten notes:*
- Redistribution of Blood
 - Tachy Cardia.
 - α Receptor
 - Brain

endothelium

بالذات في
Lung → V.C
→ Aldosterone

میں نے اس کے
وہ میری طرف سے ہے۔
عصمت علی
B. 7-7

Regional Control by $\text{CO}_2 \uparrow \rightarrow \text{V}_0 \downarrow$

Distribution of
Blood
 750 mL
 Tachy
 Cardia.
 α Receptor
 Skin.
 β Receptor
 heart.

Wounds

Definition

- Disruption of continuity of any body structure by physical injury.

Classification of wounds

They are classified into:

1. Closed (blunt): e.g. motor car accident, falling from height.
2. Open (penetrating):
 - a) Accidental e.g. gunshot, cut wounds, stabbing, bites
 - b) Surgical trauma.

According to extent of tissue injury:

	Tidy wounds	Untidy wound
Mechanism of injury	Incision	Crushing or avulsion
Cleanliness	Clean	Contaminated
Tissues	Healthy	Devitalized
Tissue loss	No or little	Much tissue loss
Healing	1 st intention	2 nd intention

It can be classified according to onset into:

- i- Acute wounds
- ii- Chronic wounds

1. Acute Wounds

I- Open Wounds:

- Surgical: Caused by sharp instruments. They are always tidy and cleanly cut.
- Non-surgical.

II- Closed Wounds: with an intact epithelial cover

I- Open wound

1. Abrasions

Etiology:

- Friction with a rough blunt hard object, leading to scraping away of the superficial layers of the skin.

Clinical picture:

- Very painful due to exposure of sensitive nerve endings.

Treatment:

- Cleaning with antiseptics and non-adherent dressing.

2. Penetrating (puncture) Wounds

- Etiology: pressure by sharp object (pointed instrument).

Characters:

- a) The wound depth is greater than its length on the skin surface increasing the risk of injury of deep important structures.
- b) External opening is small and drainage is poor increasing incidence of infection.

- Class.: acc. to (etiology, extent, onset)
- C/P: pain, swelling, comp.
- Comp.: -general: shock, inf., crush s.
- local: inf., inj., comp. of healing
- Invest.: ATLS invest.
- ITT: 1st, 2nd, definitive

Importance:

- Can miss injured viscus by unaware surgeon by suturing.

3. Bites

Etiology:

- Human bites
- Animal bites

Characters:

- They are lacerated & contaminated wounds with increase incidence of infection. "Mixed wound"

4. Incised (Cut) wounds

- Etiology: Incising by a sharp cutting object.

Characters:

- a) The cut edges are cleanly cut.
- b) It is longer than deeper.
- c) No much tissue destruction.
- d) There is usually extensive hemorrhage.
- e) Tendons and nerves are liable to be cut.

5. Lacerations

- Etiology: Severe violence with heavy objects.

Characters:

- a) The wounds are severely traumatized and devascularized.
- b) Irregular in shape.
- c) Usually contaminated, so the risk of infection is high.
- d) Inflammatory edema will develop after a few hours and will raise tension inside the wound to a high level leading to 2nd ischemia of the tissues.

6. Degloving

Etiology:

- a- Open degloving: e.g. ring avulsion injury with loss of finger skin
- b- Closed degloving: e.g. rollover injury caused by passage of motor vehicle over a limb.

- Character: skin and subcutaneous fat are stripped by avulsion from its underlying fascia, leaving underlying structures exposed.

7. Missile wounds

- Very serious, as the bullets transmits high kinetic energy to the tissues. Kinetic energy of the missile is determined mainly by:
 - a. Its velocity (V).
 - b. Its weight (M).
 Depending on this formula: $MV^2/2g$ ($g = \text{gravity}$).
- May be high velocity missile injuries (rifles) or low velocity ones (pistols). Since velocity is included in the formula it proves that high velocity missiles are more dangerous than low velocity missiles.
- The damage is due to:
 1. Direct damage by the missile in its track.
 2. Shock waves: sometimes damage occurs in areas faraway from the missile track.
 3. Temporary cavitation effect.
 4. In the missile strikes a bone, the fragments of shattered bone act as 2nd missiles producing more damage.

فرد في بطنه
تلف
Peritoneum
↓
تسحق
الطحال

عقبة
حساسة في
رجله

لطفه يتي - طاهرة دي داخله مسخنة

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الطقة بتي - طاهرة دي داخله سخته

غزة في بطنه
تطرح
Penitance
↓
تسقط
بالطول

عقبت
حساف في
رجله

Closed wound

1. Contusion

- **Etiology:** blunt object producing bleeding into the tissues.
- **Character:** skin discoloration (therefore is called Bruises or Ecchymosis).
- **Fate:** takes variable time for the discoloration to clear off (needs no specific treatment).

2. Hematoma

- **Definition:** Collection of blood in a potential space
- **Clinical picture:**
 - a. A tender cystic swelling.
 - b. In deep parts may be invisible (e.g. in thigh and gluteal region)
- **Complications:**
 - a- Infection → abscess.
 - b- Fibrosis → firm mass.
 - c- Calcification → hard mass.

- **Treatment:** Aspiration using a large bore needle or open surgical evacuation.

2. Chronic Wounds

- These are wounds that failed to undergo the usual sequence of acute healing within reasonable time (usually 3 months).
- This is due to one or a combination of factors affecting wound healing.
- Chronic wounds are arrested at the inflammatory & proliferative phases of wound healing, with overgrowth of granulation tissue with minimal wound contraction & collagen formation.
- Managements of chronic wounds is composed of:
 - a. Management of general factors.
 - b. Local wound management.
- **Examples of chronic wounds are:**

1. Leg Ulcers

See ulcers

2. Pressure Sores

See plastic surgery

3. Diabetic foot

Treatment

• Management of patient at ER:

- I. Follow ATLS protocol in management of polytraumatized patient (see surgical trauma).
- II. Prophylaxis against tetanus.
- III. Prophylactic antibiotics are prescribed. In deep or lacerated wounds, antibiotics against anaerobic infections should be added.
- IV. If there is bleeding from the wound, the best way is to stop it by direct local compression. (Don't apply a tourniquet except as a temporary measure before taking the patient to the theater).
- V. For Bone injuries: If a fracture is suspected arrange for a plain X-ray and splint the fracture.
- VI. For arterial injuries: Feel the distal pulses, if not palpable reassess after treatment of shock. Urgent duplex is mandatory if the pulses are not palpable.

apple

Medial aspect of thigh

اول مرة ← تاجه

نعم بها ← مياه سحيه

antibiotic +

Chronic leg ulcer

• Management of wound in the theater:

- I. Cleaning of wound with saline irrigation.
- II. Removal of any foreign body (very important because it is a media for micro-organisms)
- III. Wound debridement: to leave healthy vascularized tissue.
- IV. Sterilize the wound by antiseptic (Betadine®).
- V. Inspect the wound & deal with every injured structure:
 - a) **ARTERY/VEIN:** according to general principles.
 - b) **NERVE/TENDON:**
 - ↳ Clean incised wound: repair.
 - ↳ Contaminated wound: delay repair.
 - c) **MUSCLE:**
 - ↳ Necrotic/Ischemic muscle: it is dark-red or grey in color, does not contract on pinching & does not bleed → these are completely excised.
 - ↳ Clean incised muscle: can be repaired by mattress sutures.
 - d) **FRACTURED BONES:** If there is any possibility of infection → external fixation.
 - e) **FASCIA:** If wound is contaminated or there is extensive tissue destruction → leave the fascia open.
 - f) **SKIN:**
 - ↳ Clean & early (< 6 hrs) → 1ry closure.
 - ↳ Contaminated or late (> 6 hrs) → delay 1ry closure (3-4 days)

• All missile injuries require exploration.

• In contaminated wounds:

1. Don't do nerve repair.
2. Don't do tendon repair.
3. Don't close the deep fascia.
4. Don't close the skin.

Wound Healing

Definition

- An organized & complex cascade of cellular & biochemical events that results in restoring the integrity of the wound.

Components

- a) Wound contraction.
- b) Granulation tissue formation.
- c) Epithelialization.

- **Types:** 1ry, 2ry, 3ry intention
- **Etiology:** general, local factors
- **Stages:** ① inflammatory ② proliferative ③ remodeling
- **Comp.:** -Wound (failure, infection)
 - Scar.
 - Contracture

Inflammatory Response to injuries.

قطع قطع طلع

Good European
Brings
More Blood.نام عليه
بساله اسود

HCP

نظريه
الرقاع
ن

Types

	1 st intention	2 nd intention	3 rd intention
Character of wound	- Tidy wounds (clean) - Opposed edges - No complications	- Untidy wounds - Unopposed edges (d.t. hematoma or infection).. - Infected	- Contaminated wounds initially left open for 5 days, if no infection closed by delayed 1 st suturing
Scar	- Nice & neat - Minimal fibrosis. - Strong scar	- Ugly. - Extensive fibrosis (d.t. filling with granulation tissue) - Weak scar.	As primary
Duration	- Seals in 1-2 days - Heals in 1-2 weeks	Much more time is Needed	Closed when the wound is healthy to avoid bacterial contamination

Stages of Wound Healing

They are 3 phases overlapping each others:

1. Hemostasis & Inflammatory Phase

- It takes about 5 days, however it may be prolonged in infection.
- Starts by blood vessel injury → aggregation of platelets to subendothelial collagen & activation of collagen cascade → $VD \rightarrow$ ترشح → Fluid + Cellular element.
- From platelets, cytokines & growth factors are released → stimulates chemotaxis of leucocytes, macrophages & lymphocytes.

Macrophages:

- Plays an important role in phagocytosis & wound debridement.
- It releases further growth factors → activates fibroblasts & endothelial cells.

2. Proliferative Phase

- It is composed of proliferation of:

	1. FIBROBLAST	2. ENDOTHELIAL CELLS	3. EPITHELIAL CELLS
Source	Surrounding tissue	Intact venules	Wound edges & injured epithelium
Action	Secrete collagen fibers	Form new capillary buds	Basal-cell of epidermis undergo hypertrophy & mitosis & start migration to close the defect
	Type III + Hucopolysaccharide Forms granulation tissue		

Proline Type III

Vit C

hydroxyproline Type I

- In large open wounds the epidermis grows for few cm & then slow-down & a raw area persist.
- Young epithelium is thinner than normal epithelium & has **No sebaceous or sweat glands nor hair follicles.**

Tensile strength

Maturation

No Tensile strength Proliferative.

3. Maturation & Remodeling phase

- Deposition of collagen:
 - 1st collagen III then after weeks collagen III decreases & collagen I increases.
- Remodeling:
 - Fibers become lined up along the stress lines of the wound, with increase in its tensile strength, but never attain its original strength.
 - The process continues for about 1 yr.

Factors Affecting Wound Healing

A- General Factors

- Type of patient:
 - Age: old patients have poor wound healing d.t. reduced rate of protein turnover.
- Malnutrition.
 - Protein deficiency leads to diminished synthesis of collagen & ground substance.
 - Vitamin C deficiency leads to failure of collagen maturation.
 - Vitamin A deficiency leads to deficient epithelization.
 - Calcium, Zinc, Copper & Manganese also affect wound healing.
- Medications:
 - Steroids, cancer chemotherapy & immunosuppressive drugs inhibit wound healing.
- Debilitating diseases as uremia, jaundice diabetes, cirrhosis & malignancy delay wound healing.

B- Local Factors

1- Type of wound (tidy Vs untidy):

- Mechanism (incision Vs crushing).
- Environment (dry Vs moist*, temperature, pH, infection).
- Tissue loss.

- Vascularity:** a good blood supply (e.g. in the face & scalp leads to nice healing while in a poor blood supply (wounds below the knee) causes delayed healing.
- Irradiation:** impairs wound contraction & granulation tissue formation as it causes ischemia due to EAO. endarteritis obliterans.
- Immobilization:** helps healing, because movement damage the blood supply of granulation tissue.
- Tension:** any increased tension in the wound will lead to ischemia & impaired healing. Sutures under tension, hematoma and infection increase tension inside the wound.
- Infection:** delays healing. Fibroblasts compete with bacteria for O_2 & nutrition. Moreover, bacteria secrete collagenolytic enzymes, which destroy collagen fibers.

- Foreign bodies & necrotic tissue impair wound healing.
- Adhesion of the wound to a bony surface prevents wound contraction (chronic venous ulcers, wounds on chin of tibia).
- Impaired venous drainage, as in post-phlebotic limbs, impairs wound healing

venous Pressure ++

General

Type P+

Condition of P+

Medication

Local

Type P wound

of wound

Infection

Irradiation

Immobilization

F.B

Bl supply

X Tension

Factors which favor wound infection include:

1. Presence of foreign bodies.
 2. Dead tissues. → ++ anaerobic Bacteria.
 3. Ischemia. → HCO
 4. Suturing under tension. → ischemia
- After 1 week → the wound has only 3% of its final strength.
- After 3 weeks → the wound has 20% of its final strength.
- After 3 months → the wound has 80% of its original strength.
- The tissues never regain their original tensile strength.

N.B.: Dry Vs Moist wound healing

DRY WOUND HEALING

- Hard to epithelialize.
- No wound nutrition.
- Scabs delay healing due to desiccation of underlying tissues.
- Dead tissues are a good media for anaerobic bacteria.

MOIST WOUND HEALING

- Easy to epithelialize fast.
- Allows wound nutrition.
- Prevents scab formation.
- Allows granulation through cell migration.

Dead space

Fluid زرع

infection

HCO

Complications of wound healing

1. **Wound failure (wound dehiscence):** can be a result of any general or local factor affecting wound healing. (Burst abdomen)
2. **Stretching of the scar**
3. **Hypertrophied scar:** → Preventable
 - The scar is raised above the surface but it remains within the confines of the wound within months it may regress.
 - This problem is common in the shoulder and the pre-sternal area.
4. **Keloid formation: (non preventable)**
 - There's over activity of the healing process leading to excessive scar tissue which is raised above the surface and extends beyond the confines of the original wound. It can follow burns, traumatic or surgical wounds, ear holing & vaccination. HCO
 - Persons with dark skin are more prone to keloid formation and there's a familial predisposition. Common at ear lobules, shoulder and pre-sternal areas
 - Treatment of hypertrophic scar and keloids:
 - a. Continuous pressure by silicone gel sheets → causes ischemia of the small blood vessels leading to diminished activity of the fibroblasts
 - b. Intralesional steroids. Triamcinolone & a local anesthetic are injected in the dermal region of the scar.
 - c. Surgical excision: Recurrence rate after simple surgical excision may reach 80%. To minimize recurrence intramarginal excision of the scar is recommended with intraoperative excision of steroids
5. **Contracture:**
 - This is a pathological shortening of the scar tissue resulting in deformity in scar overlying joints
 - Proper positioning of the joint during healing can minimize the deformity
6. **Surgical site infection**

"haemovac"

Suction Drain

Normal Healing in Specific Tissues

A- Nerve Healing:

1. Distal to wound, Wallerian degeneration occurs.
2. Proximal to wound, traumatic degeneration occurs till last node of Ranvier
3. Regeneration occurs, from proximal to distal directed by neurotrophism at a rate of 1 mm / day.
4. Overgrowth and /or poor approximation may lead to neuroma formation.

B- Tendon healing

Same phases as wound healing but differs in that:

1. Two main mechanisms for delivering nutrients and other blood elements to tendon:
 - a- **Intrinsic:** consists of vincular blood flow and synovial effusion.
 - b- **Extrinsic:** depends on the formation of fibrous adhesions between tendon and tendon sheath
2. Early active mobilization: prevents adhesions limiting range of motions, but in 1st 3-6 weeks the tendon lacks tensile strength, so it must be protected by splintage in this period to avoid rupture of the repair.

Suture Materials

→ See operative book.

Wound Dressings

- It is used to provide the ideal environment for wound healing & prevent bacterial overgrowth.

Common forms of wound dressings

A. Occlusive dressings:

- Conventional gauze & adhesive plaster → prevention of evaporation that prevents undue dryness & protection.
- Local hypoxia of the wound → stimulates capillary proliferation & favors autolysis of sloughs.
- It is non-suitable for infected wounds & highly exudative wounds.

B. Semiocclusive dressings:

- Permeable to water vapor & gases.
- Impermeable to bacteria.

C. Alginates dressings:

- Formed of seaweeds → create a moist environment by formation of fibrous gel on contact with wound exudates.

D. Osmotic agents:

- Absorbes exudate, e.g. honey, urea crystals & Debrisan

Factors that enhance whole healing reactions

1. Vacuum assisted closure (VAC) → afford -ve pressure → enhances formation of granulation tissue & wound contraction. "dead space"
2. Increase local hydration of the wound → afford moist environment (moist healing)

early active mobilization
protection

Bursal fluid

خمرة واحدة
قبل كده
عالي
في ال
5 Marks

لوفى حديد كثير
غير على الجرح مرتين
في اليوم

الحديد
التي
تعملها

غالي

اعشاب
شكرية

البيكتريا متكويش

عشان ينشف الماء

ميتيل البواقي من
Glycerine
mg
Sulfate

Osmotic

Wounds

Definition :

It is forcible loss of continuity of soft tissues by mechanical trauma.

Classification :

[A] **Open wounds** : associated with loss of the continuity of the skin.

[B] **Closed wounds** : associated with intact skin.

[A] **Open wounds** : Are either incised, stab, lacerated or gunshot.

1. **Abrasion** : Partial denusion of superficial layers of skin due to friction of skin with a rough surface. → *أشهر جرح الخلية*

2. **Incised wounds** : due to sharp objects are liable for more bleeding & less liable for infection.

3. **Stab or punctured wounds** : is caused by pointed objects as nails and daggers. *character → small external wound with liability of Deep injuries*

4. **Lacerated wounds** :

- They are caused by blunt heavy objects.
- These wounds are **less liable for profuse bleeding** (as the blood vessels are crushed).
- **More liable for infection** whether nonspecific or specific as tetanus & gas gangrene. *High risk of Infection*

5. **Missile wounds** :

- They have an inlet (for entry of the bullet) and may have an exit.
- The damage is due to the following factors :

- (a) Direct damage by missile track.
- (b) Shock waves preceding missile injury to the body.
- (c) Temporary cavitation effect.
- (d) If bone affection; bone fragments act as secondary missiles.

• The degree of injury by the missile depends on its size and velocity :
(a) **Low velocity missile** : e.g. bullet from a pistol produces injury due to its direct effect by laceration and crushing within the passage of the missile.

(b) **High velocity missile** : Tissue necrosis occurs several centimeters on either side of the track due to the cavitation effect. It is more lethal.

6. **Bites** : either human or animal bites. They are very liable to infection.

[B] Closed wounds :

1. **Contusion = Ecchymosis** : It is extravasation of blood from injured small blood vessels with no formation of a swelling.

2. **Hematoma** : Localized collection of blood in the loose tissue spaces.

↳ Swelling

Complications of wounds

General complications :

1. **Shock** : Neurogenic, hypovolemic or septic shock. *Pain*

2. **Crush syndrome** :

Etiology :

- Prolonged ischemia with irreversible muscle necrosis.
- Toxic substances from damaged muscles leak out into circulation & are excreted in urine.
- Acid myohematin precipitates in the tubules producing anuria & irreversible renal failure.

Treatment :

(a) **Prevention of renal failure** : By alkalanization of urine, blood transfusion, IV fluids & mannitol. *as Myoglobin is Acidic*

(b) **Care of the injured limb** :

- i. Should be immobilized & kept cool. *عقد و ما تحس كبروش*
- ii. Care of the wounds.

Local complications :

- 1. Injury to important structures.
- 3. Retained foreign bodies.

- 2. Infection.
- 4. Complications of healing.

Management of open wounds

I- First aid treatment :

1. Control bleeding by compression.
2. Protect the wound from contamination.
3. Analgesics for pain.
4. Splintage of fracture if the wound is associated with it i.e. compound fracture.

II- Definitive treatment :

- Do 3 anti : (anti-shock, anti-biotics and anti-tetanic serum).

1. Clean cut (incised wounds) within the first 6 hours :

- No contamination and no risk of infection.
- The edges of wound are approximated by suture.
- Tendons and nerves can be sutured. This aims at healing by primary intention

الوحيد الذي يصلح فيه
Nerve & Tendon

2. Lacerated wound within the first 6 hours :

- The edges of the wound are excised and primary suture of the wound is done.
- Tendons and nerve repair is postponed 4 - 6 weeks.

العادة =

3. Wounds seen within 24 hours and are potentially contaminated :

- The dead muscles and dead tissues are excised, foreign bodies are removed.
- The wound is left opened for dressing with no suture for 4-6 days.
- When healthy granulation fills the wound delayed primary suture can be done.

هو جرح أدي ح التوتيل
بها تاخرها

4. Infected wounds :

- Debridement is done in which only dead and devitalized tissues are removed.
- No excision of the edges of the wound is done, as this will break the body defense against infection.
- Daily dressing is done.
- When the wound gets cleaner it can be closed by suture or by skin grafting.



5. Missile injuries :

- Incise the skin generously but do not excise much skin from the wound margin.
- Incise the deep fascia widely and identify the neurovascular bundles.
- Excise dead muscles and foreign bodies but do not remove comminuted bone. Cut tendons and nerves are not sutured.
- Leave the wound open to allow free drainage.

وما تدور في الحلقة
وتبوظ أكثر

6. Amputation :

- Indicated in severely lacerated wounds with comminution of bones and injury to the blood supply and nerves of the limb that cannot be repaired.

SKIN & MS. → Lacerated
A. & V. → Injured
N. → Damaged

N.B.:

1. If there is any doubt about the advisability of suturing the wound, do not suture. Primary suture of a contaminated lacerated wound can lead to more damage than the original injury.
2. If there is an associated fracture and there is any possibility of infection, it is advisable to stabilize the fracture by some form of external fixation, because internal fixation is contraindicated in these circumstances.

Wound healing & management

Wound healing

قرائة

Components of wound healing :

1. **Wound contraction** : helps to diminish the size of wound.
2. **Granulation tissue formation** : which is replaced later by **fibrous tissue**.
3. **Epithelialization**.

Stages of wound healing :

مستبعد جدا نظريا

1. Hemostasis & inflammation phase :

- Injury of blood vessels leads to aggregation of platelets & activation of coagulation.
- This is followed by **chemotaxis** of leucocytes, macrophages & lymphocytes.
- **Macrophages** play an important role in phagocytosis & wound debridement.
- This phase **lasts for about 5 days**, but it may be prolonged if there is wound infection.

2. Proliferation phase :

①

→ Type 3 collagen

- This phase is characterized by the proliferation of **fibroblasts** that secrete collagen fibers.
- **Endothelial cells with fibroblasts** form the granulation tissue. ②
- **Epithelial cells proliferation**. "Epithelialization" ③

3. Maturation & remodeling phase :

- **Deposition of the collagen fibers become thicker** and they get **arranged along the lines of stress** leading to increase in the tensile strength of the wound.
- The process of remodeling continues **for about one year**.

In addition to the above mechanisms, **wound contraction** helps to diminish the size of wound.

Types of wound healing : هام شفوي

1. Healing by primary intention : This occurs in clean wounds when they are immediately closed by sutures or clips. Healing occurs with minimal scarring leading to a nice scar.

2. Healing by secondary intention : This occurs when the wound edges are not approximated or when gaping occurs as a result of a hematoma or wound infection. Healing occurs by infilling with granulation tissue and so there is more fibrous tissue. The resulting scar is ugly.

الجرح اعمق من 2cm. ←

3. Healing by tertiary intention : potentially contaminated wounds are left open for about 5 days. At the end of this period if there are no signs of infection; delayed primary sutures can be performed.

Intention of the surgeon

1vy	2vy	3vy
نية الجراح	نجد ما نقل	الجراح غير نية
منه اول مرة	رجع تاني	عشان عايز اظلم
يقفل	فالجراح غير	شكل الجرح خلط
	نية تاني	
	(يارب شغل)	
	وقلاص	

* Factors affecting wound healing

عوامل جراحية

I- General factors :

- 1. Age :** Wound healing is slow in elderly persons due to a reduced rate of protein turnover.
- 2. Nutritional state :**
 - (a) **Protein deficiency** leads to diminished synthesis of collagen.
 - (b) **Vitamin C deficiency** is responsible for lack of maturation of collagen.
 - (c) **Vitamin A deficiency** leads to deficient epithelization.
 - (d) **Calcium, zinc, copper & manganese** also affect wound healing.
- 3. Debilitating diseases :** Uremia, jaundice, cirrhosis, diabetes & malignancy delay wound healing.
- 4. Drugs :** Steroids and cancer chemotherapy impair wound healing.

II- Local factors :

- 1. Vascularity :** A good blood supply (e.g. in the face & scalp) leads to nice healing while a poor blood supply (wounds below the knee) causes delayed healing.
- 2. Immobilization :** Movement delays healing.
- 3. Local irradiation :** Inhibits wound contraction & granulation tissue formation.
- 4. Tension :** Any increased tension in the wound will lead to ischemia & impaired healing. (علاقة التوتر بالشفاء)
- 5. Infection :** Fibroblasts compete with bacteria for oxygen & nutrition. Moreover, bacteria secrete collagenolytic enzymes, which destroy collagen fibers.
- 6. Foreign bodies & necrotic tissue :** impair wound healing.
- 7. Adhesion of wound to a bony surface :** Prevents wound contraction e.g. chronic venous ulcers.
- 8. Impaired venous drainage :** as in postphlebotic limbs impairs wound healing.

* Complications of healing :

عوامل جراحية

- 1. Wound failure (wound dehiscence) :** General or local factors which affect wound healing can lead to wound failure. Failure of an abdominal wound is called burst abdomen.

الخراج يفتح

2. Stretching of the scar.

Items	3. Hypertrophic scar	4. Keloid
Definition	<u>Delayed</u> maturation of collagen fibers in a wound.	<u>Failure of maturation</u> of collagen fibers in a wound.
Induration	Limited to the scar.	<u>Extends</u> beyond the scar.
Course	Stationary after 6 months. <u>ثابت بعد 6 شهور</u>	<u>Progressive</u> even after 6 months.
Sites	Common in shoulder & presternal area.	Ear lobules, shoulder, presternal area.

Incidence of Keloid :

Black persons are more prone to keloid formation and there is a familial predisposition.

Treatment of hypertrophic scars and keloids :

- (a) Continuous pressure by silicone gel sheets.
- (b) Intralesional corticosteroids.
- (c) Surgical excision : Recurrence rate after simple excision may reach 80%. To minimize recurrence intramarginal excision of the scar is recommended together with intraoperative injection of steroids.

5. Contracture :

resulting in deformities.

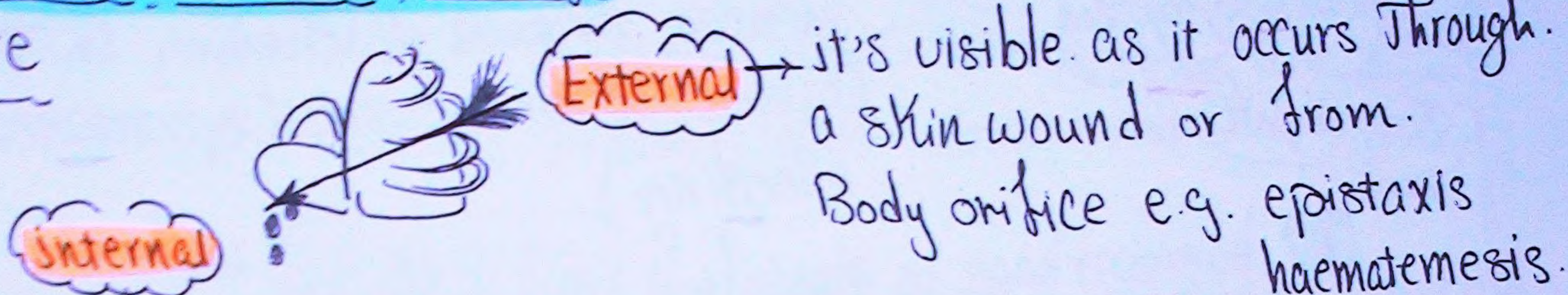
6. Wound infection.

Acute Haemorrhage & Blood Transfusion.

Loss of Bl. volume.
Loss of O₂ Carrying Capacity

Classification of haemorrhage.

1 Site



Interstitial

Bleeding into Tissue.
Forming a haematoma.

~ fracture haematoma

into a **Body Cavity** as haemothorax. haemoperitonium.

4 → **acute**.
→ **Chronic** haemorrhoids.

2 Type of Ruptured vessel



Artery

Bright Red

Pulsatile Jets

more from proximal than distal.

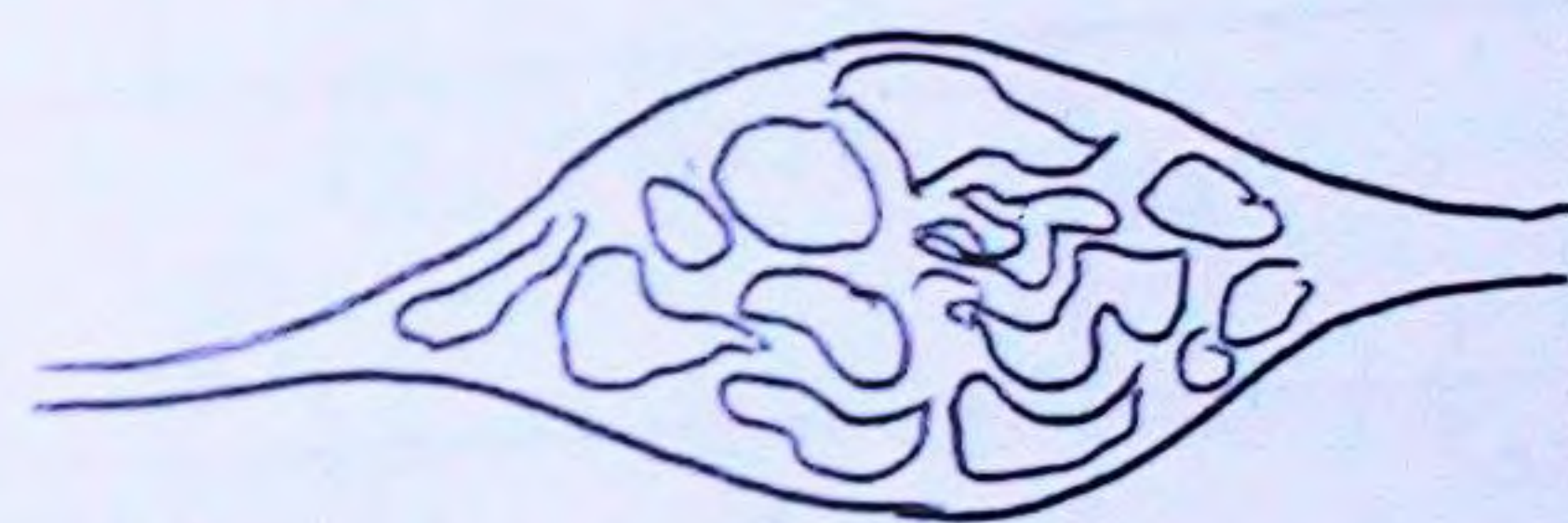


vein

Dark red

steady flow

more from distal than proximal.

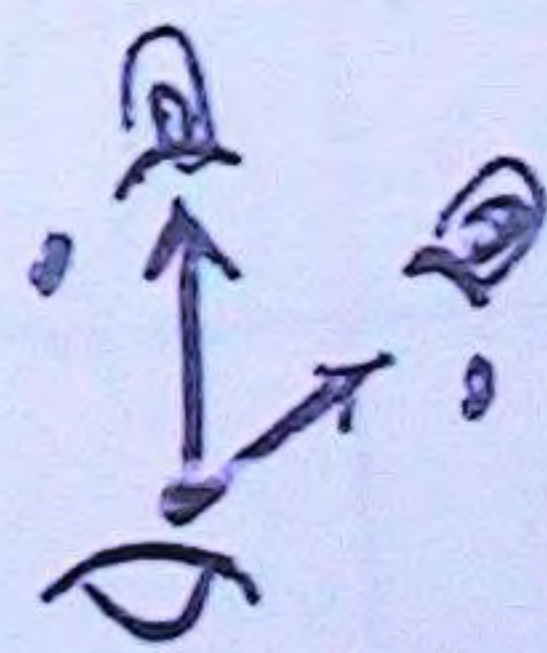


Capillary

Bright Red

Diffuse ooze

بيخفت
مياه حمرة



3 Timing in Relation to the onset of Trauma.

1ry at the Time of injury.

Reactionary within **24 Hrs** Correction of hypovolemia or 2ry to Post-operative pain → Dislodge of a clot or slipping of insecure Ligature.

2ry **1-2 w** infection eroding Bl. v may be fatal "Carotids"

After sloughing of the skin following Radical neck Dissection.



مشی باخفت زده الانسداد همی ری الجلی بوقفه با

- Traumatic. accidental, surgical, interventional
- Pathological - atherosclerosis (Ruptured aortic aneurysm)
- inflammatory (Bleeding peptic ulcer)
- neoplastic (haematuria in Renal Cancer)
- Bleeding Diathesis

HCO Thrombocytopenia
(خبر عامه)
Coagulopathy

- increase amount of Bleeding in previous Causes or may Cause (Spontaneous Bleeding)

5 haemorrhage is classified into 4 classes.

Normal amount of Bl. in adults 70 ml/kg
children 80 ml/kg

Parameter	class I	class II	class III	class IV
Bl. Loss	upto 15% 750 ml	15 - 30% 750 - 1500 ml	30 - 40% 1500 - 2000 ml	> 40% 2000 ml
mental status	normal to anxious	anxious to restless	aggressive to drowsy	drowsy to unconscious
Skin	normal	pale & cold	paler, cooler	pale & very cold
Capillary Refill	Normal	> 2 sec	> 2 sec	> 2 sec undetectable
Pulse/min	< 100	100 - 120 *	120 - 140 *	> 140
Systolic BP	N	N (supine) *	Low *	Low
Diastolic	N	high	Low	Low
Pulse P.	N	Low	Low	Low
Resp. Rate	14 - 20	20 - 30	30 - 35	> 35
urine output	> 30 ml/hr	20 - 30 ml/h	10 - 20	0 - 10

[2]

Multorgan failure

- for c/p - Pathophysiology Treatment Refer to eka
- (hypovolemic shock)

HCOs Let's

Estimating The amount of Fluid Loss.

1. Clinical Data (الجدول السابق)
2. Type of injury
 - Closed fracture Tibia 500 - 1500 ml **HCO**
 - fracture femur 500 - 2000 ml **HCO**
 - fracture pelvis 2000 - 3000 ml **HCO**

3. Blood Loss in operation

= Blood in suction Device + Blood mopped up by swabs

Correction factor x (وزن المريض - وزن الجراح)
Depending on (1.5 - 2)
The Magnitude of the operation.

4. During operation: Clot at site of closed fist = 500 cc

Investigations

- CBC
- Coagulation profile.
- Cross matching.

- Site of Trauma.
- Complication
- Organ profile

haematocrit

NoB initially

Normal Bl & plasma loss in same proportion

4 - 6 h. later reduced due to (hemo-dilution)

why!!!!

① Movement of part of the interstitial fluid to the Circulation.

② Fluid Replacement Therapy

* Patient on warfarin immediate antidote is **FFP**

* Vit K start to act 1 - 2 Days later

[3]

Transfusion of Blood & its Components

1 - Collection & storage

400 - 450 ml. Blood

70 - 100 ml of
• Anti Coagulant

ACD
AL
Better Citrate
or CPD + 1

2 - 6 °C During storage

(Frozen Blood)

no coagulation factors n
no platelets.

stored at : (-80 - -180 °C

press Blood → Remove plasma.
Then add Glycerol to
the red cells.

2 Blood components

* indications

precaution

Whole Blood

Class III, IV haemorrhage

ABO & Rh.

Packed RBC's

anemic, elderly, Cardiac patient as it
++ O₂ Carrying capacity without vol. overload

ABO & Rh

Fresh Frozen Plasma

- non specific coagulation factor ↓
- Coumarin over dose (No platelets)

ABO
- 40 °C

Fresh plasma

after separation of RBC's rich in

platelets & Co

Platelets Concentrate

1st or 2nd Thrombocytopenia.

1 unit of Blood ++ platelets by

ABO
10,000 - 15,000
Freshly prepar

Cryoprecipitate Factor VIII & Fibrinogen

Bleeding with Fibrinogen Depletion.

on at

Factor VIII

haemophilia A for 8h only in blood.

Factor IX

haemophilia B, Coumarin overdose

Albumen

5% - 20%

hypo albuminemia.

4

No B it's no longer indicated to use fresh Blood to correct Coagulopathy The specific deficient component should be used.

* 3 Complications of BT Transfusion

Intro: Though generally safe & simple, the procedure of Blood Transfusion is not free of complications. The majority of these are simple, yet some are fatal others produce chronic serious illness as hepatitis, AIDS. Attention to strict rules of Donation & administration of Blood reduces these complications.

in The Donor

- neurogenic shock.
- anemia
- Local thrombophlebitis

in The Recipient

- 1 immunological
 - incompatible RBC's lead to acute haemolytic reaction.
 - incompatible WBC's pyogenic.
 - incompatible platelets purpura.
 - Reaction to protein in plasma allergic reaction.

acute haemolytic Reaction

Due to :- Ab in the Recipient's Blood against one or more of antigens in the Donor's cells. or plasma containing high titres of Ab against the Recipient RBC's.

Clinically :- • after Transfusion of < 50 ml :- fever chills Constricting chest pain, Dyspnea pain in flanks. + Tachycardia + hypotension. • anaesthetized patients - sudden Tachycardia, hypotension Bleeding tendency.

5

• a major haemolytic Reaction → haemoglobinuria.
Jaundice.
acute Renal failure.

• Delayed haemolytic Reaction

5-10 Days in patient immunized.
to foreign antigen by a previous
Transfusion or pregnancy.
un explained jaundice, unexplained pyrexia.

management:-

- Stop the Transfusion immediately
- Retyping & matching.
- Correct shock by IV infusion of lactated Ringer + IV Corticosteroid.

Forced.
alkaline diuresis.

- watch urine output
 - osmotic diuresis as mannitol.
 - keep alkaline urine to protect against acute tubular necrosis

IV Bicarbonate may be needed.

- if acute RF developed it must be treated

• Pyrogenic Reaction The commonest, unpleasant comp.

- Due to presence of recipient ab against the Donor's WBC's or Bacterial contamination.

• Clinically chills, fever, headache, nausea, vomiting.

- management stop the Transfusion &
Give IV aspirin or paracetamol.

• Post infusion purpura Patient previously sensitized against foreign platelets antigen.

• allergic reactions

- Due to The Recipient respond to an allergin in the Donor's Blood.
- Clinically - mild itching & urticaria
- severe Reaction with laryngeal edema & collapse.
- management - antihistaminics.
- Corticosteroids.
- if the reaction was severe stop Transfusion.

② Congestive Cardiac Failure

- in elderly patient with Rapid administration of large volume.
- C/P Dyspnea, Cough, haemoptysis, edema.
- Prevention Transfuse Packed RBC's for correction of anemia in elderly.

③ Transmission of infections

- viral hepatitis (C, B):- whole Blood - or Blood products.
now it's obligatory to test Bl. Donor against them.
Screen for CMV in immunocompromised.
- AIDS / HIV:- Blood or its Products.
- Syphilis:- (Rare) spirochaetes can't survive in Bl. Bank.
for more than 4 Days.
- Malaria:- only by RBC's Not by Blood Components
- septicemia:- Can survive But no replication in refrigerator
But if Bl. allowed to warm. Bacteria can grow
G-ve endotoxin may cause septicemic shock.

4. Hyperkalemia.

prolonged

- K^+ out to cells
- Cardiac arrest
- Cardiac arrhythmia

5. Citrate intoxication :-

Citrate binds to Ca^{+2} causes
 $\downarrow Ca^{+2}$ augments action of hype
 myocardium.

Prevention :-

for each 2 Bl. units Give
 20ml Calcium gluconate.

6. Air embolism.

7. Transfusion Related acute lung injury (TRALI) incompatibility (Donor's Ab) (Recipient)

8. Complications of massive Bl. Transfusion

• Definition Transfusion of 2500 ml at
 or 5000 ml or more over 24h.

1. Hypothermia :- may cause acidosis or arrhythmia
 so warm Bl. by Transfusing

2. hyperkalemia.

3. hypocalcemia.

4. Coagulation failure. Stored Bl. poor in
 & factor VII,

so Transfuse 1 unit of platelets & 1
 Plasma for each 1 unit of stored

5. Diminished O_2 Carrying Capacity of RBCs

9. Physiological changes occurring in stored Blood

Potassium Content

HCO

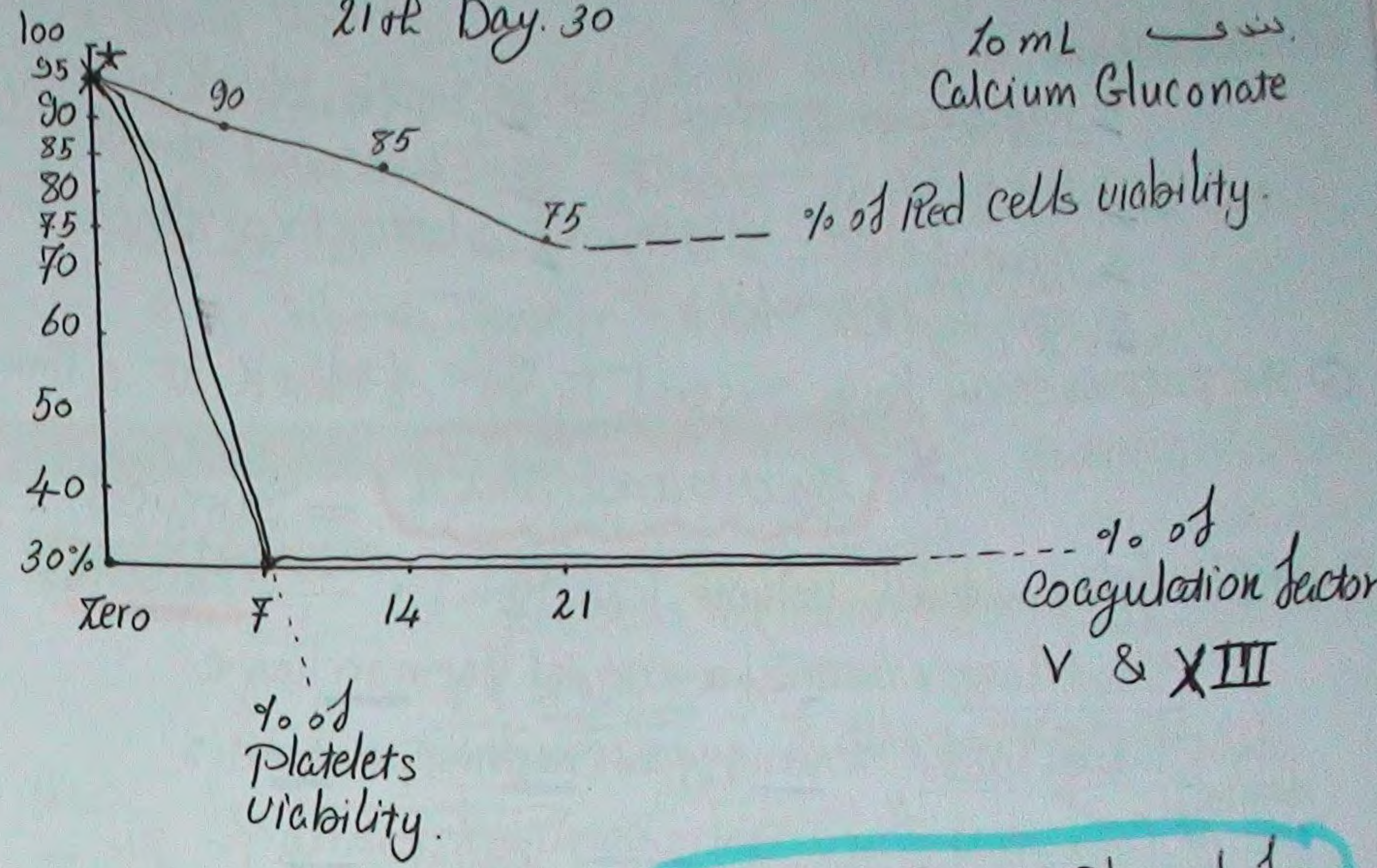
0 Day 95% 3.5

7th Day 10

14th Day 25

21st Day 30

mmol/L



Stored Blood to ml

% of Coagulation factor V & XIII

alternatives of Bl. Transfusion "how to minimize Bl. needs for (homologous Bl. Transfusion)"

1. autologous Bl. Transfusion.

• a patient who is going to have a Major elective surgery
 can Donate some of his Bl. over several days to be
 kept in Refrigerator & Transfused Back during
 surgery.

2. preservation of Bl. loss During surgery to be
 reinfused again this requires a special apparatus.
 (Cell saver)

Shock (صدمة)

Definition it's a pathophysiological condition that leads to inadequate tissue perfusion through microcirculatory changes leading to impaired cellular metabolism.

Classification

1. hypovolemic shock.
2. Cardiogenic shock.
3. Neurogenic shock.
4. anaphylactic shock.
5. septic shock.
6. Endocrinal shock.
7. obstructive shock.

one patient may have several types of shock at a time.

* hypovolemic shock = Surgical Shock.

* **Etiology** :- ↓ Bl. volume Due to ...

- a. Blood loss : internal or external haemorrhage.
- b. Plasma loss : Burns, pancreatitis, peritonitis.
- c. Na Containing fluid : severe vomiting, diarrhea, I.O, high output intestinal fistula.

Loss of 10% → as Donating a Bl. unit
 20% → you will feel sick.
 40% → you will go into hypovolemic shock.

Pathophysiological Response :-

1. stop the Bleeding.
2. Maintain effective circulatory vol. { Neural (أعصاب) / Endocrinal (أغذية وهورمونات) }
3. Microcirculatory changes.
4. Cellular Derangement
5. Acid Base imbalance.
6. effect on individual organs.

Written

Details

1. Stopping the Bleeding.

- Vasoconstriction & Retraction.
- Platelet Plug.
- Blood Clotting.

in sequence. HCO الترتيب

No. 3: This mechanism is More effective in Complete Transection of the vessel wall than a partial Tear. HCO على وسع الشح
 - More effective in Traumatic than pathological causes.
 e.g. fibrous tissue in the base of Bleeding Peptic ulcer.
 Reflex. spontaneous autoregulation.

2. Maintaining effective circulatory volume.

a. Neural factor :- * ↓ stimulation of stretch receptors in (Baroreceptors) متمنع
 * ↓ inhibitory discharge of Both aorta → Vagus & Glossopharyngeal on Carotids
 also
 ++ HR
 ++ COP
 Sympathetic The VNC

1. veins :- Constriction "Move the Bl from the capacitant vessels (2/3 vol) to the circulation".

2. arterioles :- ++ TPR. "Not uniform" as the Metabolic needs of the heart & Brain overcomes the alpha adrenergic vasoconstrictor Discharge.

3. Inotropic - Chronotropic.

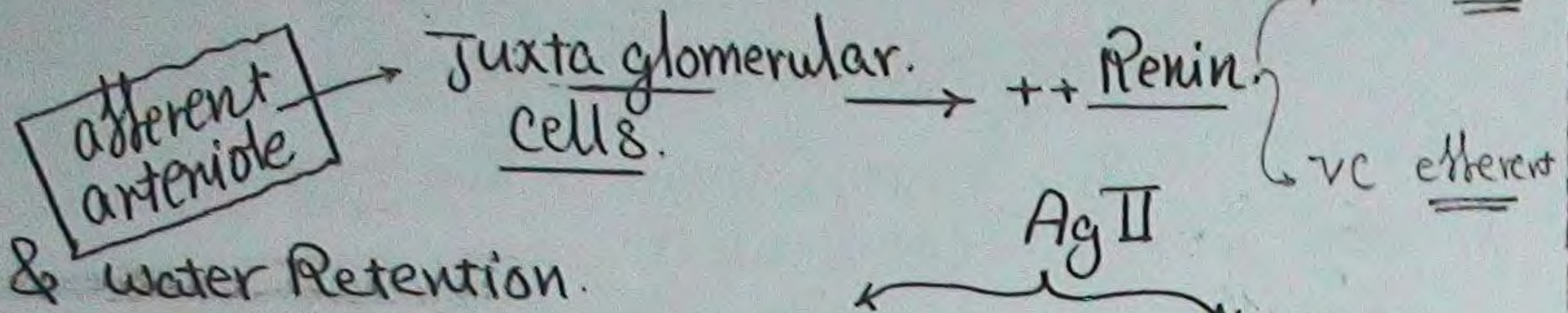
B) Endocrine Factors

1. **Catecholamines** - adrenal medulla. Autonomic Nerve endings. α, β Receptors. Redistribution of Bl. Flow. α Receptor $\rightarrow$ V.C $\uparrow$ HR $\uparrow$ force of contraction.

2. **ACTH, Cortisol, GH, Glucagon** $\uparrow$

Insulin Release $\downarrow$ by adrenaline & noradrenaline.

3. **RAAS**



Salt & water Retention.

No B Neuronal Response occurs within seconds.

Endocrine Response occurs within 20 min.

4. **ADH (vasopressin)** *

Blood Loss $> 10\%$ stimulate ADH release water reabsorption.

severe haemorrhage $\rightarrow$ high levels of ADH producing vasoconstriction.

C) Transcapillary refill

$\downarrow$ Bl. volume $\rightarrow$ constricting arterioles.

$\downarrow$ capillary hydrostatic pressure.

"shot of fluid from the interstitial to the circulation." - internal fluid Transfusion

hemodilution.

3) microcirculatory changes

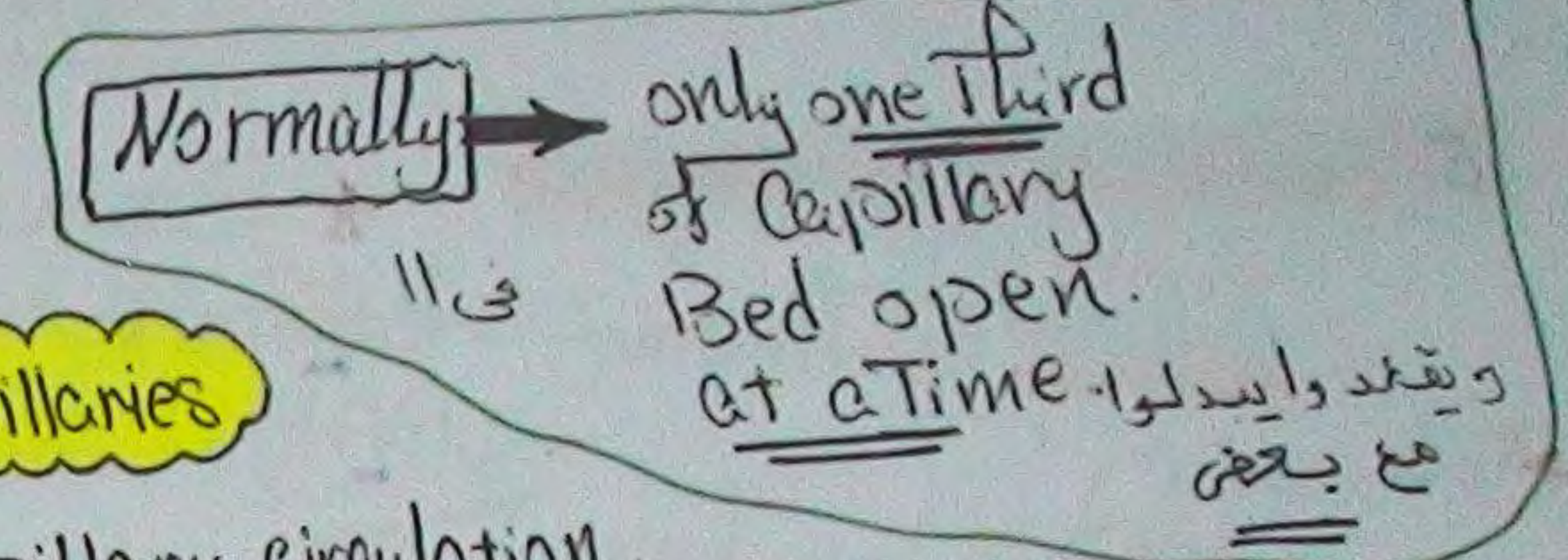
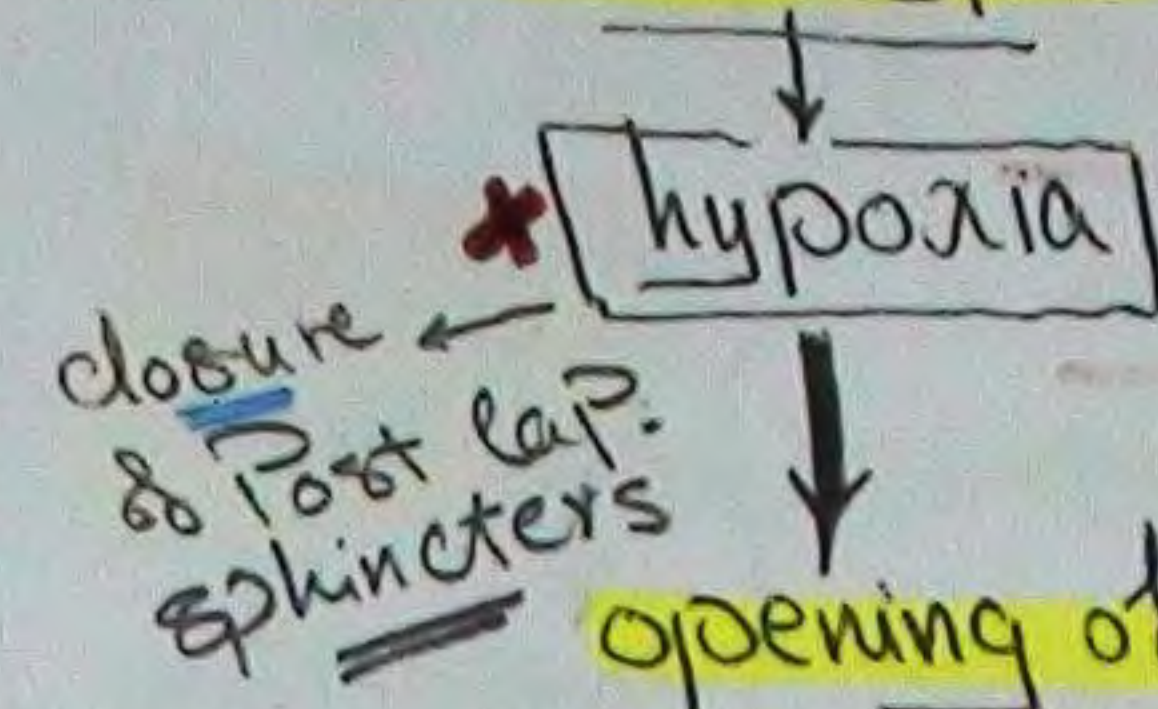
1. **Catechol amines**

Constrict the precapillary sphincter $\rightarrow$ Blood shunts to venous side.

Dilated capillary sluggish.

Circulation $\downarrow$ Pressure $\rightarrow$ Fluid shifts from (IST) $\rightarrow$ vascular compartment

2. **untreated hypovolemia**



- further slowing of capillary circulation.
- This encourages spontaneous coagulation. **DIC** (Consumption Coagulopathy)

3. **DIC + hypoxia** $\rightarrow$ (capillary-endothelium) (leakage of large protein molecule) Dragging with them $\rightarrow$ huge amount of fluid.

irreversible shock

Pre-capillary sphincters

Flush toxic metabolites to the body

ذبي الحائل والبيان

4. **cellular Derangement**

$\downarrow$ O_2 $\rightarrow$ anaerobic Metabolism

Lactic acid.

Deterioration of cell function.

Finally $\rightarrow$ lysosomal rupture cell death "Necrosis"
vital organs are spared. eventually if exposed to hypoxia $\rightarrow$ Extensive Damage.

irreversible shock

Failure of Na^+/K^+ pump.

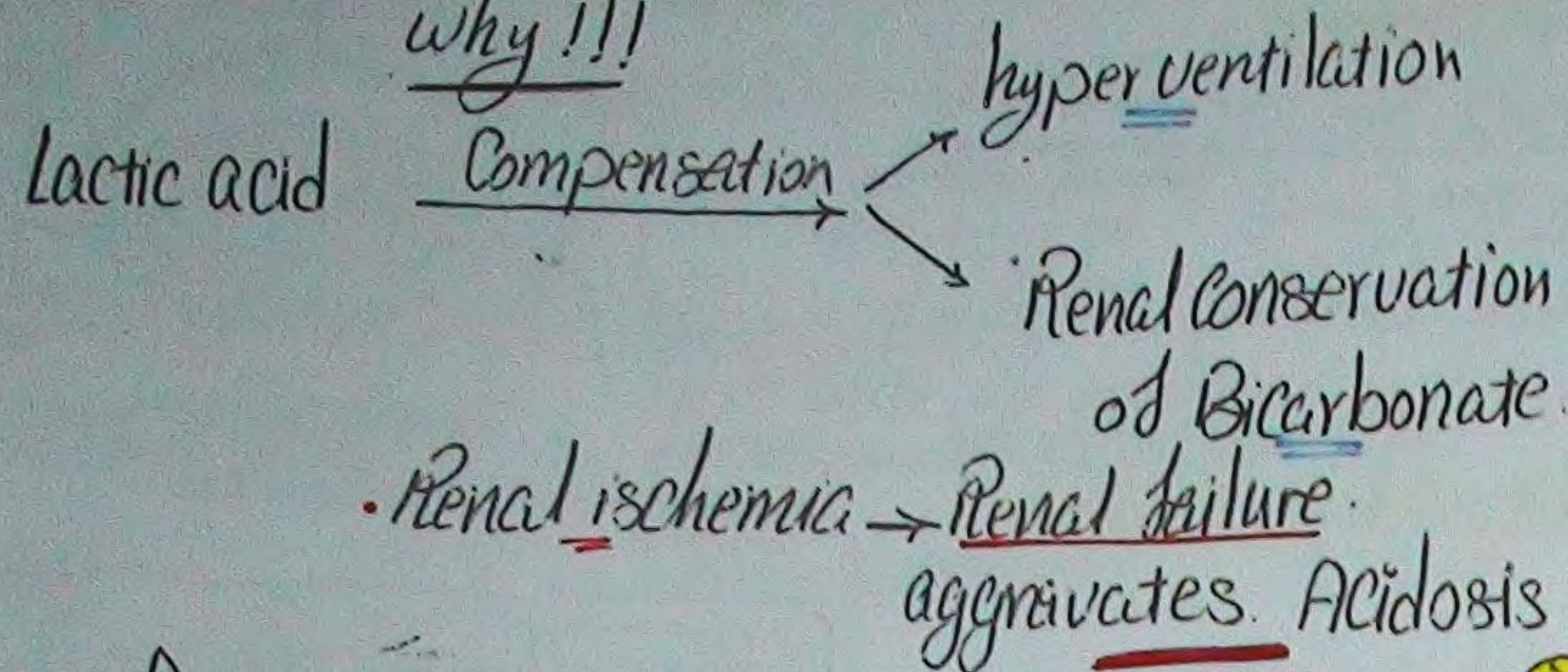
+++ Na^+ accumulation of intra cellular water.

hydropic degeneration

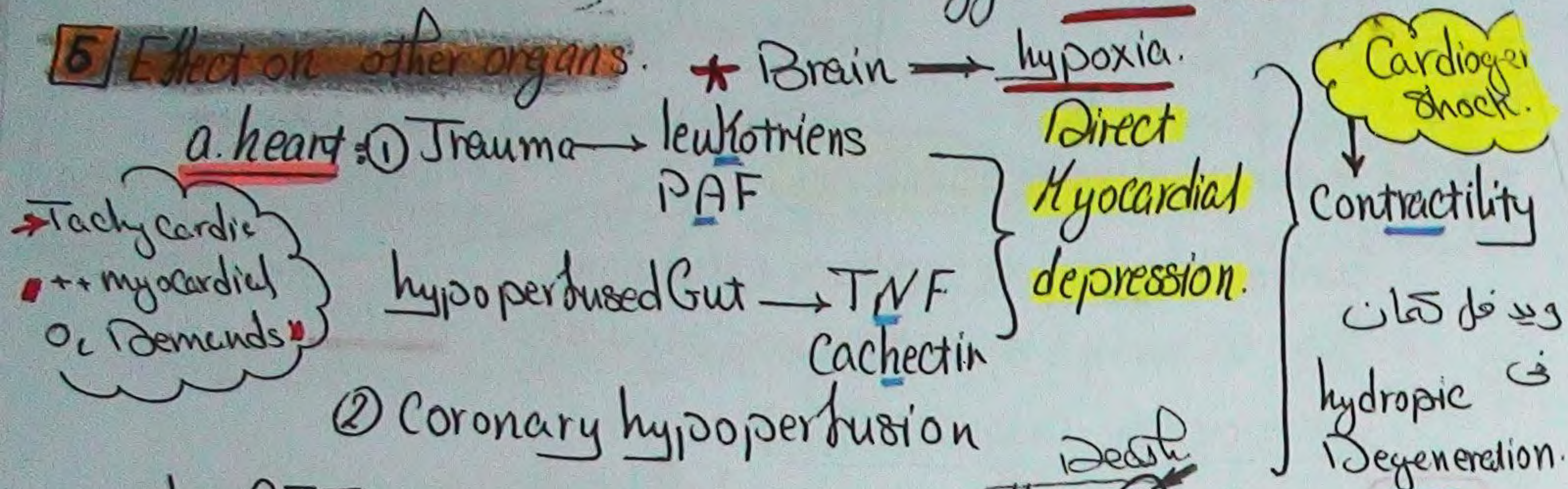
4 Acid-Base imbalance:-

Shock → metabolic acidosis

why!!!



5 Effect on other organs:



b. GIT

ischemia → stress ulcer

Cortisol → ulcerogenic

α-Receptors

weak colonic mucosa → Translocation of Bacteria & endotoxins to circulation.

Septic shock

"Death after resuscitation in a previously shocked Patient"

c. Liver ischemic hepatic Dysfunction.

d. Kidney: ↓ RBF → Tubular Necrosis (Reversible) → Cortical Necrosis (Irreversible)

BL shift from Cortex to Medulla

RAAS ↓ GFR → ++ Tubular Reabsorption of salts

prolonged hypotension → acute Tubular Necrosis

e. Lung

ARDS "non cardiogenic pulmonary edema"

3 Clinical picture

No B₂ Loss of 1500 mL in healthy individuals May not lower BL pressure (initially)

a Symptoms:

1. weakness & fainting esp. with standing.
2. hypotension in a healthy person means loss of at least 1500 mL.
3. hypotension in "Coronary artery Disease Patient" means loss of 500 mL.
4. Patient is Cold & Thirsty.

b Signs:-

1. mental status: (Anxious to Drowsy) But Remain alert
2. pulse & BL pressure:-
 - (mild BL loss < 500 mL) → compensated → Normal
 - more BL loss → Tachycardia with Normal BL P
 - more BL loss → hypotension
3. ↓ PP Thready pulse
4. RR Tachypnea & air hunger ← acidosis
5. Temperature → hypothermia → Better avoided
6. Pale skin → Cold (v.c) "slow capillary refill & Collapsed veins"

7. Oliguria Results due to diminished renal perfusion.

Stages of shock

- ① Compensated with loss > 150% it will fail.
- ② Decompensated > 40% loss.
- ③ Refractory. Failure of vital organ & Death.

- If the patient rapidly treated with adequate replacement the vital signs will return back to normal

C.P → MOSF ← ك. ت. م. س

Nº13 Ischemic perfusion Syndrome

- hypoperfusion + hypoxia + Metabolites & inflammatory mediators.
- once normal perfusion is restored. flushing of these mediators to the circulation. → more endothelial damage.

(irreversible shock)

Def: Complete vascular collapse with hypotension → unresponsive to fluids, drugs. Leading to lethal CVS & Cardiac Dysfunction.

Related to:

1. Duration & vol. of haemorrhage
2. age
3. pre existing cardiac damage.
4. presence of Massive Trauma.

Before reaching the conclusion that the shock is irreversible 1st exclude other causes of failure to respond to treatment

- ① Continuous unsuspected Bl. loss
 - ② inadequate vol. Replacement ↓ CVP.
 - ③ occult Thoracic injury → Cardiac tamponade.
 - ④ acute Myocardial insufficiency.
 - ⑤ Tension pneumothorax
- } ↑ CVP

* management of hypovolemic shock - haemorrhage

1. stop haemorrhage :- "if the cause was haemorrhage" written

a. 1st aid measures

- packing, pressure, position.
- skin wound :- Cover by dressing & apply pressure → manually 4 min
→ sphygmomanometer
→ bandage.

The key is to control the cause.

- elevation of the limb above the heart stop venous bleeding ↓ arterial. ي. ل. س. د. ١٥
- pneumatic anti shock garment (PASG) ++ TRR → ↑ BP
Can stop L.L, pelvic, abdominal haemorrhage.
- Ballon Tamponade in Bleeding varices.

b. operation

- 1ry → Control Bleeding point by ligation or Diathermy
- Reactionary → open the wound & control the Bleeding vessel
- 2ry - The wound is packed with antibiotics & sterile dressings.
- systemic antibiotics
- if Bleeding didn't stop → open the wound Drain infection expose Bleeding vessel "ligate"

2. Resuscitating BL Volume.

a. Venous access in u.l. or by venous cut down Saphenous.

withdraw BL for Cross matching

b. The volume & Type of solution used Dep. upon ¹ Class of haemorrhage & whether it has an ² active Bleeding or not

Class II • The Deficit is estimated (15-30%)
• Lactated Ringer - amount $3 \times$ Deficit
• administration:-

عيار سيزف
أديك دلتز
Ringer lactate
و نبتت ابيله

- 1st 1-2 Liters as a bolus with
This is highly effective monitoring pressure.

if responding → Continue slowly → then maintain

if not Responding why?? → CVP

Class III & IV or continuous haemorrhage

- as Class II But needs Blood Transfusion
- initial vol. is the estimated deficit (1500)
- The available BL → Cross matching.
Clean BL in the Thorax.
Aspirated & Transfuse

* Continue Transfusion till haematocrite reaches
urine out 50 ml/h.

CVP risen to upper half of the Normal Range

in absence of Blood use Colloid solutions

Burn → Plasma.
I.O → Crystalloids

Plasma
albumin
Dextran
artificial
Substit

3. Optimizing O₂ Delivery

40% O₂ in class II

100% O₂ in class III & IV

"Then adjust according to ABG"

→ Resp. failure → IV intubation + mechanical ventilation

Dopamine B → inotropes, vasopressors. May be used.
Dopamine B → if raising O₂ carrying capacity didn't improve O₂ Delivery.

4. Monitoring

a Patient Responding to 2L of Ringer lactate clinical parameters are enough.

ECG for shock induced arrhythmia.

- pulse - BP - venous filling
Capillary perfusion.

Serial Rectal Temperature

Urine output optimum 0.5-1 ml/Kg/h.

b Invasive monitoring procedures old patient Class III & IV
↓ Cardiac reserve

① CVP • End Diastolic pressure "preload" in Rt Chamber.

Catheter → Median Cubital vein.
Subclavian vein.
IJV.

Check position by CXR. rule out pneumothorax

• Normally 5-10 cm water.

if the heart is normal CVP will reflect Blood volume.

② Pulmonary artery wedge pressure. (PAWP)

- Swan Ganz catheter.
- To a small Branch of the pulmonary artery
- The pressure ~~let~~ detected by the catheter reflects that on left side of the heart

Packed RBC's

③ ABG.

④ Blood Lactate.

⑤ haematocrite & Hb. → > 30% of Normal

C ECG

D Temperature.

Difference Between peripheral & Core Temp.

normally Core is higher but should not exceed 2°C .

(any increase of this Gradient → ↓ perfusion)

normally
- Core temp. → $37,5$
- Peripheral skin temp. → $36,5$
الفرق 1

4 Positioning - elevate legs with the Rest of the Body in supine position.

5 pain Relief

- early immobilization of a fracture.
- IV analgesic.
- Sedation "asph is Irritable"

{ poorly perfused. }
{ S.C or Ms. }
ما تديره مش

6 Inotropic agents.

(Dopamine, Dobutamine) ++ Renal Bl. Flow.
++ Urine output
++ Myocardial Contraction.

More details

- Crystalloids may be the only fluids given to patients with hypovolemia caused by loss of water & electrolytes.
- Glucose 5% is not used in shock** because glucose is rapidly redistributed, so ↑ amount of ECF (without electrolytes) → dilutional hyponatremia → ↑ ICF (brain edema & ↑ ICT).

Complications of venous catheter:

- Hemothorax or pneumothorax.
- Arrhythmia.
- Sepsis.
- Pleural injury.
- Thrombosis.
- Air embolism.

2- Septic Shock "Endotoxic Shock"

Etiology

Causative organisms

- Gram -ve bacilli "The COMMONEST"
- Staphylococci.
- Candida. → in AIDS patient

Source of infection

Common sources are:

- Peritonitis: caused by perforated viscus, gangrenous bowel or leaking anastomosis.
- Cholangitis or genitourinary infections.
- Infected central venous catheter.

Predisposing factors (low immunity)

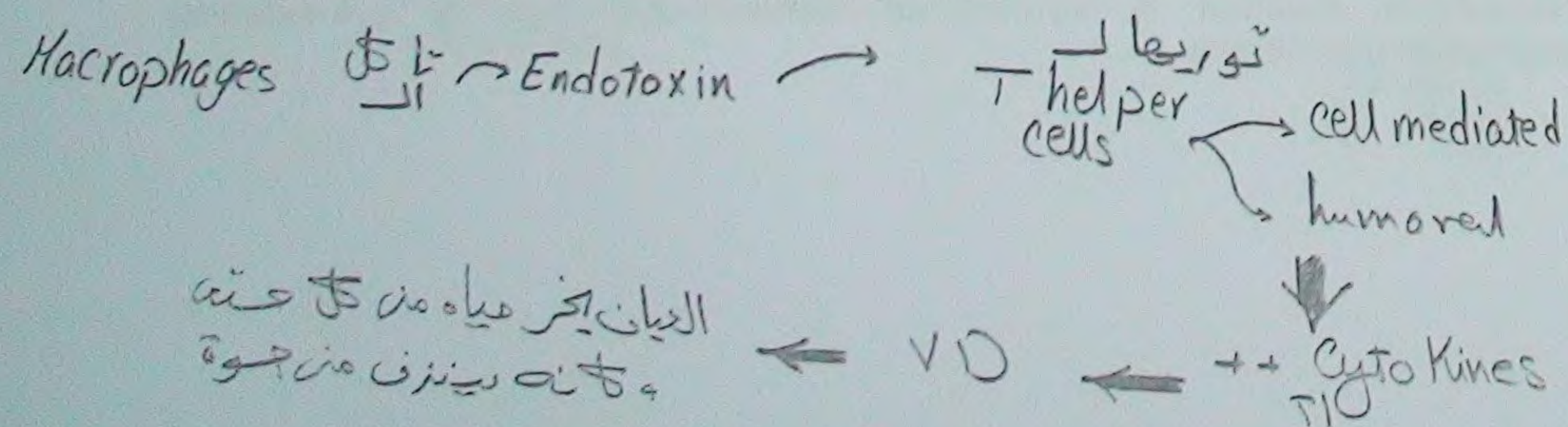
- Old age.
- Malignancy, malnutrition.
- Chemotherapy, corticosteroids or immunosuppressants.
- Diabetes mellitus.
- HIV/AIDS.

The incidence of septicemia and septic shock are rising although the presence of more powerful antibiotics which may be attributed to:

- Developing reservoirs of resistant and virulent organisms.
- Concentration of infected patients in Critical care units.
- More extensive operations in elderly and poor risk patients.
- Salvage of severely injured patients.
- Immunosuppression by organ transplantation and chemotherapy.

Pathophysiology

- Dead bacteria → liberate lipopolysaccharide from the cell wall of gram -ve bacilli. (endotoxin)
- Endotoxins trigger complex immunological reactions which are: macrophages & Kupffer cells in the liver → release cytokines in large amounts → harmful effect on microcirculation particularly on capillary endothelium.



Microcirculation:

- Maldistribution of cardiac output. Under effect of cytokines arteries undergo vasodilatation & AV shunts open results in:
 - ↓ PR, which is increased by nitric acid.
 - Bypass of capillaries where oxygenated blood passes from arterioles to venules.
 - Impairment of oxygen delivery to tissues.
- DIC, which is a result of:
 - Sluggish capillary circulation.
 - The action of platelet activation factor.
- Vascular endothelial damage under the effect of cytokines. This results in leakage of protein rich fluid from the circulation into the interstitial space causing edema.

Cellular derangement: (sick cell syndrome)

- O₂ delivery is reduced because of:
 - Maldistribution of blood.
 - Late pump failure.
 - Hypovolemia.
- O₂ uptake & utilization by the cells is also directly suppressed by the toxins.

Individual systems & organs:

Lungs:

- ARDS is a common sequel. "non cardiogenic pulmonary edema"
- Generalized capillary endothelial damage → alveolar & interstitial edema, the results are:
 - ↓ compliance & alveoli filled with fluid → both affect ventilation.
 - Opening of arterio-venular shunts → divert blood away from capillaries → impairs perfusion.
 - Thickened alveolo-capillary membrane (i.e. interstitial edema widens the space between blood in capillaries & air in alveoli) → impairs gas diffusion.
- So, the 3 components of respiration are impaired leading to respiratory failure.

- Brain:** If blood pressure drops below 60 mmHg → cerebral ischemia.

Liver:

- ICU jaundice (under effect of ischemia & chemical mediators → cholestasis & hyperbilirubinemia may develop)

GIT: ischemia of the gut mucosa produces:

- Gastro-duodenal stress ulceration.
- Gut barrier failure → translocation of bacteria & endotoxins from colon to blood stream with further deterioration of the condition.

Cytokines:

- Cytokines are large peptides.
- Its normal function is communication to mediate a useful inflammatory response.
- These are:
 - Tumor necrosis factor.
 - Interlukines
 - Platelet activation factor.
 - Prostaglandins (prostacyclin & thromboxane).
 - Nitric acid.
- In large amounts they produce harmful effect on microcirculation

* **Clinical Picture*** **Clinical picture of septic shock:**

- Described under 2 stages: due to the major difference in outcome according to stage of diagnosis:

1. **Hyper-dynamic "warm" septic shock (Early phase):** **SIRS**

- Diagnosis is Difficult and high index of suspicion is required to detect it at early stage.

- Restless & confusion.
- Skin: flushed, warm & dry extremities.

- Vital data:

- Fever $>38^{\circ}\text{C}$ + Chills.
- Mild decrease in ABP.
- Tachycardia.
- Tachypnea.

- High cardiac output.

- Oliguria.

2. **Hypo-dynamic "cold" septic shock (Late phase):**

- If treatment is not effective or diagnosis is late the patient passes to cold phase.

- Picture similar to hypovolemic shock with reduced cardiac output:

- Skin: cold clammy.

- Vital data:

- SBP $< 90\text{mmHg}$
- Tachycardia.
- Tachypnea.

- Oliguria.

- Multi-organ failure starts at this stage.

SIRS

→ hyperdynamic phase of Septic Shock.

- SIRS is a global inflammatory state that yields a particular set of symptoms & objective findings before sepsis & shock set in.

- There are 4 SIRS criteria, presence of 2 or more indicates SIRS:

- Temp. $> 38^{\circ}\text{C}$ or $< 36^{\circ}\text{C}$.
- HR > 90 beat/min.
- Tachypnea > 20 breath/min or $\text{pCO}_2 < 32$ mmHg.
- WBCs > 12000 or $< 4000 / \text{mm}^3$.

normal:- $4000 - 11000 / \text{mm}^3$

- 2 criteria or more = **SIRS**.

- 2 criteria or more + source of infection = **sepsis**.

- 2 criteria or more + source of infection + organ dysfunction = **severe sepsis**.

- 2 criteria or more + source of infection + organ dysfunction + hypotension = **septic shock**.

Diagnosis is helped by

- ↑ level of lactate in blood.
- Leucocytosis.
- Looking for a source of sepsis.
- Repeated blood culture.

* **Complications & causes of death: (80% mortality)**• **Multi-organ failure:**

- ARDS.
- Acute renal failure.
- Hepatic dysfunction.

• **DIC.**

- Acute erosive gastritis "stress ulcer".

Investigations

- Assessment of general condition:** (should be done serially for follow up)
 - CBC: Marked leucocytosis (or leucopenia, late) & thrombocytopenia (degree of infection & associated complications).
 - ABG: PO_2 , PCO_2 , pH (hypoxia & hypercapnia in ARDS).
 - Electrolytes & Blood sugar (for dehydration).

• **For the cause & source:**

- Isolation of organisms from source of infection & blood by:
 - Cultures: should be done on anaerobic & aerobic media.
 - Blood cultures.
- Location of septic focus:
 - X-Ray: Abdomen & Chest.
 - U/S & CT scan.

• **For complications:**

- KFTs & LFTs. (MOF)
- ECG monitoring. (IHDs)
- Coagulation profile. (DIC) → Fibrinogen Degradation Products.

Treatment

- Treatment should be started as soon as possible.
- Patient is admitted to ICU.
- The most important components are:
 - Supporting body systems.
 - Fighting infections.
- Monitoring is essential for guidance of treatment.

a) **Resuscitation:**a- **Circulatory support:**

- This is the initial priority in managing septic shock to maintain mean arterial pressure to keep the patient alive.
- Fluid replacement: till CVP is 10-12 cm H_2O or pulmonary wedge pressure 12 - 15 mmHg.
 - By Ringer's Lactate.
 - If low Hct. → packed RBCs or whole blood transfusion.

N.B.: huge amounts of fluid are often needed exceeding 10 liters in few hours

2- Drugs:

- Inotropes & vasopressors: dopamine & dobutamine are given if the patient remains hypotensive despite adequate fluid replacement.
- Start by nor-epinephrine or dopamine to keep a mean blood pressure above 65 mmHg.
- If no response, epinephrine or dobutamine is prescribed.

Respiratory support:

- O_2 by mask.
- If $\text{pO}_2 < 60$ mm Hg → mechanical ventilation.

b- **Renal support:**

- Adequate circulatory support improves renal blood flow.
- Hemodialysis is required in acute renal failure, until the kidneys recover.

c- **DIC:** Fresh frozen plasma.b) **Fighting infection:**a- **Eradication of sepsis:** e. g.

- Drainage of huge abscess or peritonitis.
- Resection of gangrenous bowel.

→ Treatment of the Cause

General surgery

b- Antibiotics:

- Parental, combined, broad spectrum started early without waiting for culture & sensitivity results (3rd generation cephalosporin + Amnioglycosides + Metronidazole).
- Then changed according to culture & sensitivity. (Garamine)

c) Continuous monitoring:

- 1- Vital signs (temperature, pulse, BP and RR) and ECG.
- 2- Urine output.
- 3- ABGs, repeated blood culture, CBC, coagulation profile & organ profile.
- 4- CVP, arterial line and pulmonary wedge pressure.

d) Strict control of blood sugar has been proved to increase survival.e) Prophylaxis against DVT and stress ulcers.▪ Corticosteroids (A point of controversy):

- Some say that it can be administered to stabilize cell membrane & antagonize inflammatory mediators.
- Others say that it's not indicated in treatment of septic shock.

E.coli:

- Normal intestinal flora.
- Commonest organism in gram -ve septicemia.
- Commonest organism in abdominal infection.

Prognosis

- High mortality ranges from 25 % up to 90% due to late diagnosis & late treatment.

3- Other Types of Shock**A. Cardiogenic Shock****Definition**

- Inadequate blood flow to vital organs due to inadequate cardiac output despite of normal blood volume.

Causes

- These are the commonest causes leading to sudden collapse:
 - Acute myocardial infarction "The COMMONEST"
 - Severe arrhythmias.
 - Massive pulmonary embolism.
 - Cardiac tamponade "this is due to penetrating wounds of the chest".
 - Myocarditis.
 - High spinal anesthesia "can cause paralysis of the sympathetic supply of the heart".

(Congested neck veins & high CVP)

HCO

Clinical Picturea) Of shock:▪ Symptoms:

1. Weakness and fainting specially when standing.
2. The patient feels cold and thirsty.

▪ Signs:

1. Altered mental status (may vary from anxious to drowsy d.t. ↓ cerebral perfusion, but the patient usually remains alert).

2. Pulse & blood pressure: "see classes of hemorrhage"
 - Loss < 500 ml → pulse & blood pressure remains normal.
 - More blood loss → tachycardia & normal blood pressure.
 - Further blood loss → tachycardia & hypotension.
3. Pulse pressure → ↓ leading to thready pulse.
4. Hypothermia, predisposing to coagulopathy & should be avoided.
5. Tachypnea and air hunger.
6. Skin becomes pale, cold (VC) and clammy with slow capillary refill (> 2 sec.) and collapsed veins.
7. Oliguria and later on renal failure d.t. ↓ renal perfusion.

b) **Of the cause:** Chest pain.

c) **Characterized by** congested neck veins & High CVP.

Treatment

- O₂ Inhalation
- TTT Of the cause:
 - M.I. → early thrombolytic therapy & potent analgesics
 - Control arrhythmias.
 - Relief of cardiac tamponade by emergency insertion of needle to drain blood in the pericardium followed by definitive surgical haemostasis.
 - Mechanical supports:
 - The intra-aortic balloon device serve the following:
 - ☞ Elevating diastolic blood pressure → better filling of coronary arteries.
 - ☞ Reduction of myocardial work.

B. Distributive shock

a- Neurogenic shock:

- There is paralysis of vasomotor fibers leading to peripheral pooling of blood & inadequate venous return. *Sympathetic*
- It is due to either:

1. Spinal shock.

► Causes:

- Complication of high spinal or deep general anesthesia.
- High transection of spinal cord due to fracture spine.

► Pathophysiology:

- Due to loss of sympathetic outflow leading to sudden severe vasodilatation.

2. Vasovagal attack.

- This is the simplest type of neurogenic shock.

► Causes:

- Severe pain (Trauma to a trigger area as a blow to testis or larynx)
- Excitement.
- Hearing bad news
- Watching bad news.

► Pathophysiology:

- Extensive VD of splanchnic arteries → sudden reduction in peripheral resistance → sudden fall in blood supply of vital organs.
- Excessive vagal stimulation of the heart → bradycardia.

N.B.: - Recovery is assisted by fainting & falling to ground that help in venous return
 - Atropine prevents this reflex but doesn't treat it.

انصراف شوك
Spinal Cord Fixation

*Hypotension
& Bradycardia*

دافى
دشلول
مبيتر فنتش

Ringer Lactate → Buffer → Salt of weak acid.

226

General surgery

• Treatment of neurogenic shock:

- I) Lie the patient flat & elevate his legs.
- II) IV crystalloid solution as ringer lactate.
- III) Vasopressors may be given.

b- Anaphylactic shock

Causes

- May follow administration of penicillin, sera or dextrans & anesthetics.

Pathophysiology

- Antigen-antibody "IgE" reaction leads to release of large amounts histamine that leads to:
 - Bronchospasm.
 - Laryngeal edema.
 - Massive vasodilatation.
 - Respiratory distress.
 - Hypotension.

Treatment

- IV crystalloid infusion.
- IV hydrocortisone.
- Antihistaminics.
- Endotracheal intubation "may be needed if laryngeal edema & stridor develops".

c- Endocrinal shock

Causes • Diabetic Ketoacidosis - Myxedema Crisis

- Adreno-medullary insufficiency:
 - After adrenalectomy for pheochromocytoma without pre-operative preparation by α & β blockers → sudden withdrawal of high level of catecholamines from the blood.
- Adreno-cortical insufficiency:
 - After bilateral adrenalectomy.
 - After removal of cortical tumors
 - After operation on a patient who has had cortisone therapy.
 - Those with Addison's disease under stress, infections, operations → Addisonian crisis. So, those patients should be nursed in ICU.

Pathophysiology

- Adrenal insufficiency + stress → failure to release steroids → failure to cope → severe shock.

Treatment

- Saline infusion.
- IV Hydrocortisone.

C. Obstructive shock

Causes

- 1) Tension pneumothorax.
- 2) Cardiac tamponade.
- 3) Massive pulmonary embolism.

Pathophysiology

- Reduced filling of Rt. side of the heart.

Clinical picture

- Characteristic finding is low COP & high jugular venous pressure & CVP.

Treatment

- Specific treatment of the cause otherwise failure will occur.

1st exposure.
Ab formation IgE
on mast cells

- 2nd exposure.
Bind of Ag to
sensitized. Mast cells.
Degranulation.

- It is advised for a husband not to donate blood to his wife during her childbearing period. If, after the blood transfusion, the woman develops an antibody to an antigen on the father's red blood cells, and the subsequently born fetus inherits the father's red cell antigen, the antibody from the mother may enter the bloodstream of the fetus causing destruction of fetal red blood cells. This may cause serious anemia in the fetus and excessive jaundice in the infant after birth.

Blood Matching

Direct Matching

Drop of **patient** serum + drop of donor RBCs before transfusion → compatibility is confirmed when no agglutination occurs.

Indirect Matching

Used mainly to determine the blood group.

Blood Transfusion

Definition

- Blood transfusion is the process of transferring of blood or blood-based products from one person into circulatory system of another.

➤ **Indications:** acc. to component (whole blood, blood derivatives)
 ➤ **Comp.:** Immunological, Infection
 Stored **blood**, massive **blood**
Cannula (thrombophlebitis, air embolism)

Collection & storage of blood

- Previously it was collected on acid citrate (anticoagulant) dextrose solute (ACD).
- A better anticoagulant is citrate phosphate dextrose.
- One blood bag:
 - 70 – 100 ml anticoagulant (citrate).
 - 400 – 450 ml of blood.
- Blood must be stored at 2 – 6°C during storage.
- Frozen blood:** plasma is removed from freshly collected blood, glycerol is added. The frozen blood doesn't have coagulation factors, platelets or white cells

Blood products & Indications

- Whole blood:** (storage life 2 – 5 weeks)
 - Acute hemorrhage external or internal.
 - Operative and post-operative replacement.
 - Severe burn.
 - Intestinal strangulation.

5. Raising the resistance to sepsis.
6. Exchange transfusion in cases of erythroblastosis fetalis.

II. Blood derivatives :

- a. **Packed RBCs:** (storage life 2 – 5 weeks)
 - Severe anemia & elderly (to avoid volume overload and disease transmission).
- b. **Leucocytes**
 - Severe leucopenia and agranulocytosis.
- c. **Platelets:** (storage life 2 – 5 days)
 - 1st or 2nd thrombocytopenia and platelet dysfunction (if bleeding or undergoing surgery).
- d. **Plasma:** (storage life 1 – 2 years)
 - **Fresh frozen plasma:**
 - Stored at 30° – 40°C.
 - **Contains:** • Igs.
 - Abs (anti A, anti B Abs, so compatibility is very important.
 - Platelets & coagulation factors.
 - Burn, malnutrition and coagulopathic bleeding e.g. hemophilia, LCF or warfarin overdose (high incidence of blood diseases transmission).
 - **Cryoprecipitate:**
 - Stored at – 40°C.
 - Mainly in hemophilia, DIC & Von Willbrand disease (contains factor VIII & fibrinogen).
 - **Clotting factors concentrates:** According to the deficient factor, e.g. VIII, IX, XI.
 - **Fibrinogen:** hypofibrinogenemia & DIC.
 - **Immunoglobulin:** passive vaccination e.g. anti-tetanic serum.
 - **Human albumin:** hypoproteinemia.

Changes that occurs during blood storage

	0 days	7 days	14 days	21 days
% of red cell viability	95%	90%	85%	75%
% of platelet viability	95%	0%	0%	0%
% of coagulation factor V & XIII	95%	30%	30%	30%
Potassium content (mmol/L)	3.5	10	25	30

Routes of Blood Administration

1. **Intravenous transfusion:** method of choice.
2. Sinus transfusion (rare): mainly in infants via sagittal sinus.
3. Intra-arterial transfusion: life saving in emergency via the radial artery.

Complications

> **Immunological complications:**

1. Incompatible white cells → pyrogenic reaction
2. Incompatible red cells → acute hemolytic reaction
3. Incompatible platelets → purpura
4. Reaction to protein in plasma → allergic reaction

1. Pyrogenic reaction (commonest)

- Due presence of recipient antibodies against some components of donors WBCs
- Patient develops chills, fever, N&V & headache
- Stop the transfusion + give aspirin or paracetamol.

2. Hemolytic reaction

- **Etiology**: incompatible blood transfusion → destruction of donor RBCs by the recipient Ab.
- **Clinical picture**
 - Clinically hemolytic reactions present after 50 ml by fever, chills, constricting pain in the chest, dyspnea & pain in the flanks.
 - A major hemolytic reaction will lead to hemoglobinuria, jaundice & ARF
 - Delayed hemolytic reaction may occur 5-10 days after transfusion in patients who have been immunized to a foreign antigen by a previous transfusion or pregnancy, it presents by unexplained pyrexia or jaundice.

➤ **If patient is under anesthesia or comatose**

1. Bleeding tendency (oozing of blood, 1st) (the most important sign).
2. Progressive unexplained hypotension.
3. Unexplained tachycardia.

- **Treatment of hemolytic reaction**

1. Immediately stop the transfusion & repeat patient's blood typing & matching.
2. IV fluid (ringer lactate & IV corticosteroids).
3. Alkalinization of urine by NaHCO₃ (40 mEq).
4. Mannitol 20% 100 ml (**forced alkaline diuresis**).
5. Monitor the urine output and vital data.

3. Post transfusion **purpura** may develop in patients who have been previously sensitized to foreign platelet antigen

4. Allergic reaction:

- Ranging from urticarial patches up to laryngeal edema.
- TTT: - Antihistaminics & cortisone.
- If reaction is severe → stop transfusion.

5. Infection

- a. Viral → CMV (commonest), hepatitis B & C, HIV.
- b. Bacterial → brucellosis, syphilis (rare now).
- c. Parasitic → malaria (only by red cells, not by blood components).

6. Air embolism7. Thrombophlebitis at the site of cannula.8. Complication of transfusion of stored blood:

1. Hyperkalemia.
2. Acidosis.
3. Bleeding tendency

9. **Citrate intoxication**: leading to hypocalcemia. If the patient will receive more than 2 units of blood it is important to administer 10 ml of 10% of calcium gluconate for each 2 units of blood

10. **Transfusion related acute lung injury (TRALI)**: due to incompatibility between donor's Ab & recipient granulocytes. It gives clinical picture similar to ARDS.

11. Massive Transfusion

This implies the transfusion of 2500ml of blood at one time or 5000 ml or more over 24 hours.

- Hyperkalemia.
- Citrate toxicity → hypocalcaemia.
- Hyperkalemia + hypocalcaemia → cardiac arrhythmia.
- Acidosis.
- Circulatory overload (heart failure).
- Hypothermia. A special warming unit should be used to warm the blood before transfusion as hypothermia can cause acidosis or cardiac arrest.
- Coagulation failure + platelet dilution → bleeding tendency. Occurs when large volume of stored blood is used to replace blood losses as stored blood is poor in platelets and Factors V and VIII. In these conditions it's recommended to transfuse one unit of fresh frozen plasma and platelets for every unit of stored blood
- Diminished O₂ carrying capacity of stored RBCs

N. B.

- Repeated transfusions e.g. thalassemia → hemosiderosis (iron overload).
- Delayed hemolytic reaction may occur after 72 hours but within 2 weeks.
 - Include: delayed hemolysis & post-transfusion purpura.

Indications for transfusion of blood & its products

Product	Indication	Precautions	Storage life
Whole blood	Class III & IV hemorrhage	ABO & Rh	21 days
Red cell concentrates	Severe anemia	ABO & Rh	21 days
Fresh frozen plasma	- Bleeding due to non-specified coagulation factor deficiency. - Coumarin overdose	ABO	1 year at -40°C
Platelet concentrates	1ry or 2ry thrombocytopenia	ABO	24 – 72 hours
Cryoprecipitates of factor VIII & fibrinogen	Bleeding with fibrinogen depletion	ABO	1 year at -40°C
Factor IX	Hemophilia A		2 years
Albumin 5% or 20%	- Acute volume expansion. - Hypoalbuminemia.		4 years

Alternatives to homologous blood transfusion

- Autologous blood transfusion:
 - A patient who is going to have a major elective operation, can donate some units of his own blood over several days.
 - The blood is kept in the refrigerator to be given back to him during surgery.
- Preservation of the blood lost during surgery & its reinfusion to the patient. This needs a special apparatus.

How to Avoid Complications of Blood Transfusion

➤ Precautions during collection of blood:

1. Good **selection** of the donor (examination and tests to avoid AIDS & hepatitis).
2. **Sterilized** apparatus and bottle (to avoid infections and febrile reactions).

➤ Precautions during storage and preparation:

1. **Anti-coagulation:** by addition of specific formulas:
 - Previously, **ACD** (acid – citrate – dextrose) → storage for 1 week.
 - Replaced by **CPD** (citrate – phosphate – dextrose) → storage for 3 weeks.
 - Recently, **SAG-M** (saline – adenine – glucose – mannitol) → storage for 5 weeks.
2. **Storage at special refrigerator:**
 - 4°C for blood and packed RBCs.
 - - 40°C for plasma and its fractions.

➤ Precautions before transfusion:

1. Assurance of the **name** of the patient and **date** of storage.
2. **Fresh blood transfusion** (< 6 hours) is recommended in old age, liver disease, renal failure and urgent massive transfusion where the RBCs have temporary reduction to release oxygen to tissue in 24-72 hours.
3. Determination of the **blood group** (direct or indirect method) and Rh antigen.
4. **Cross matching** between the donor's and recipient's blood.

➤ Precautions during transfusion:

1. Don't leave blood outside the refrigerator for more than half an hour.
2. Don't heat the blood except in massive transfusion (using special warming devices).
3. **Use filter apparatus to prevent:**
 - Microscopic clots and debris.
 - Platelet aggregation.
 - Abnormal white cell membrane in stored blood.
4. **Selection of the site** (forearm or dorsum of the hand)
5. **Rate of flow** → 30 drops / minute allows 500 ml / 8 hours (if there is no acute loss).
6. **Give the first 100 ml slowly:** detect immediate reactions (febrile or incompatibility).
7. **Give calcium ampoule** every 100 cc blood

➤ Precautions to avoid over-transfusion: continuous monitoring of CVP.

➤ Precautions during massive transfusion to avoid bleeding tendency: monitoring of clotting should be done by thrombo-elastography in the theater or in the laboratory.

Pathogenesis of renal failure in blood transfusion

- Mismatched transfusion.
- Donor blood itself contains sufficient antibodies to hemolyse recipient cells.
- Intravascular hemolysis leads to an increase in the circulating hemoglobin.
- The hemoglobin is bound by plasma protein and haptoglobin until the amount exceeds 100 mg/100 ml → circulates in the blood → reaches the kidney.
- It causes renal tubular necrosis with subsequent acute renal failure.

Haemostasis & Bleeding disorders

Definition

- It is the arrest of bleeding from an injured blood vessel.
- It requires the combined activity;
 1. Vessel walls.
 2. Platelets.
 3. Clotting factors.
- In addition to normal haemostasis, the surgeon can control bleeding by different methods that are called surgical haemostasis.

Natural haemostasis

Primary haemostasis

- Arrest of blood within 1st few minutes → is achieved by:
 1. Vasoconstriction.
 2. Platelet plug formation (platelet adhesion & aggregation).

Secondary haemostasis

- After the 1st few minutes = coagulation → is achieved by two mechanisms

Intrinsic pathway

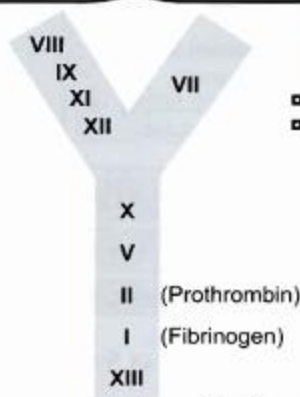
- Within few minutes
- Controlled by PTT

(Heparin mainly affects intrinsic pathway).

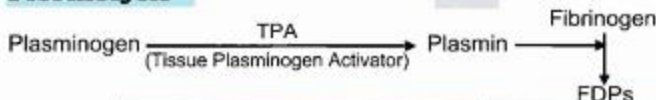
Extrinsic pathway

- Rapid within few seconds
- Controlled by PT

(Oral anticoagulants mainly affects extrinsic pathway)



Fibrinolysis



Bleeding	Platelet count
• Spontaneous bleeding	10,000 – 20,000 /L
• Bleeding with minor trauma	20,000 – 50,000 /L
• Bleeding with major trauma	50,000 – 100,000 /L
• No haematic abnormality	> 100,000 /L

➔ Inhibitors of coagulation:

1. Anti-thrombin III (activated by heparin).
2. Protein C & S.
3. Factor V (Leiden)

Coagulation screening tests

	Times	Disorders
1. Platelet count	Normal = $150 - 400 \times 10^3$	$> 100.000 =$ Thrombocytopenia
2. Bleeding time	< 8 minutes	• Thrombocytopenia. • Abnormal platelet function. • Deficiency of Von Willbrand factor • Vascular abnormality
3. PT	12 – 15 seconds	Deficiency of factor II, V, VII or X (extrinsic & common pathways) → assess oral anticoagulants
4. PTT	30 – 40 seconds	• Deficiency of factor II, V, X, VIII, IX, XI, XII (intrinsic & common pathways) → control of heparin.
5. INR	Ration between $\frac{\text{Patient PT}}{\text{Control PT}}$ Normal = 1	• Patient receiving oral anticoagulants (2 – 3) • Patient with prosthesis (2.5 – 3.5).
6. Others	• Fibrinogen. • FDPs. • Coagulation factors. • Bone marrow aspiration & biopsy → Megakaryocytes.	•

Bleeding disorders

A-Congenital disorders

I- HEMOPHILIA A & B

Definition

- It is an inherited coagulation defect (X linked recessive) due to deficiency of factors XIII → A
IX → B

Clinical picture

1. Bleeding is post-traumatic since birth (cephalhematoma).
2. Deep hematoma or recurrent hemoarthrosis.

Investigations

- Factors level is 5 – 20 % of its normal value.

Treatment

1. Infusion of deficient factors concentrated within 1 hr before surgery.
2. Fresh frozen plasma & cryoprecipitate.
3. Avoid aspirin, NSAIDs & IM injections (because large hematoma can develop).

II- VON WILLEBRAND DISEASE**Definition**

- It is an autosomal dominant disease with variable expression due to deficiency of VW factor which enhances:
 - Plasma adhesion (1ry).
 - Carrier for factor VIII (2ry).

Clinical picture

- Purpuric eruptions.
- Ecchymosis.
- Bleeding from body orifices.

Investigations

1. Prolonged both PT & PTT.
2. Deficiency of factor VIII & VW factor.

Treatment

- Infusion of deficient VW factor.

B-Acquired disorders

- They are more prevalent.

I- HEPATIC DISEASES

- Acute or chronic liver disease associated with haemostatic abnormalities

Etiology

1. Deficiency of coagulation factors, fibrinogen (PT & PTT are prolonged with ↑ INR).
2. Thrombocytopenia if associated splenomegaly or hypersplenism.
3. ↓ synthesis of inhibitors of fibrinolysis e.g. α_2 antiplatelets.

Treatment

1. Vit. K.
2. FFP (2 – 3 units).
3. Desmopressin ↑ factors VIII, VW & shorten bleeding time.
4. Antifibrinolytics or dexomercapturine.

II- VITAMIN K DEFICIENCY

- Vit. K is a cofactor for activation of factor II, VII, IX & XI

Etiology

1. Inadequate diet or TPN.
2. Prolonged use of broad spectrum antibiotics (↓ colonic bacteria).
3. Cholestatic jaundice.
4. Malabsorption.
5. Oral anticoagulants.

Clinical picture

- Easy bruising & traumatic bleeding.

Investigations

- ↑ PT

Treatment

1. Vit. K by slow infusion (5-10 mg/d /or slow IM injection 2 or 3 days [10-20 mg/day]).
2. In emergency: factor concentrates II, VII, IX, X, FFP.

III-DIC

Definition

- It is simultaneous widespread occurrence of microvascular bleeding & activation of coagulation (leading to consumption of coagulation factors) which in turn worsens the bleeding (↑ FDPs worsens the coagulopathy).

Etiology

1. Septicemia.
2. Severe shock, trauma & burns.
3. ABO incompatibility transfusion.
4. Malignancies e.g. metastatic carcinoma of the liver, pancreas & stomach.
5. Obstetric accidents (eclampsia, abruptio placenta, AF embolism).

Clinical picture

Diagnosis is suspected by

1. Diffuse bleeding from wounds, incisions, orifices.
2. Widespread bruises, purpura, mucosal bleeding.
3. Ischemic manifestations in skin & digits.

Investigations

- Low platelet count, ↑ PT, PTT, ↓ fibrinogens, ↑ FDPs.

Treatment

1. Of the cause: e.g. drainage of an abscess, antibiotics for infection.
2. FFP & cryoprecipitate or platelet transfusion.
3. Heparin is not adjusted.

IV- OTHER CAUSES

3. Anticoagulants → bleeding if not properly adjusted.
4. Massive blood transfusion.
5. Platelet disorders:
 - Thrombocytopenia e.g. ITP or 2ry.
 - Platelet dysfunction: aspirin, NSAIDs, anemia.

Preoperative evaluation of haemostasis

History

1. +ve family history of bleeding.
2. History of bleeding tendency.
3. Age of onset of bleeding (congenital or acquired).
4. Liver disease, CRF, massive blood transfusion or drug intake.
5. Criteria of bleeding:
 - 1ry haemostasis: bleeding is superficial, petichae & purpura.
 - 2ry haemostasis: bleeding is deep, hematoma, hemorrhage.
 - Excessive fibrinolysis.

Examination

- If suspecting bleeding tendency.
 1. Cutaneous signs of liver disease.
 2. Skin & m.m. for bleeding, petichae, hematoma.
 3. Cavernous hemangioma → thrombocytopenia.

Investigations

See before

- Excessive operative & postoperative bleeding is due to:
 1. Inadequate surgical haemostasis (**the commonest cause**).
 2. Hemorrhagic diathesis not detected preoperative.
 3. Acquired intraoperative coagulopathy.

Management

1. Proper surgical hemostasis.
2. Correction of hypothermia.
3. FFP, platelet transfusion unless needed.

➤ Surgical hemostasis by:

1. Ligation: a ligature tied out.
2. Under running suture.
3. Electrocautery:

a. Monopolar diathermy:

- It is called monopolar because the electric current passes through the patient body between two electrodes. One electrode (passive) is broad & is placed over the L.L. & the other (active) is usually a forceps with a fine tip which the surgeon uses to grasp the bleeding point.
- As the current passes between the electrodes, it becomes concentrated at fine tipped forceps, this generates heat which causes tissue coagulation.

b. Bipolar diathermy:

- Also the current passes between the two blades of one forceps which the surgeon uses to grasp the bleeding point.

c. Clips.

d. LASER

e. Repair of injured large vessel.

CHAPTER: 8

PRE-OPERATION & ANESTHESIA & POST-OPERATION

Pre-operation

Assessment

Objectives of pre-operative assessment

1. Improve the doctor-patient relationship.
2. Reaching **diagnosis** and making **decision**.
3. **Evaluate** the medical condition.
4. **Educate** the patient about the procedure.

Steps of pre-operative assessment

Assessment is done in 3 phases:

- I. Outpatient clinic
- II. Hospital admission
- III. Pre-operative round

History taking

When taking the history, the following should be checked:

- Cardiovascular:**
Anemia, hypertension, IHD.
- Respiratory**
Respiratory infection → should be treated first.
- Gastrointestinal**
Peptic ulcer and GERD.
- Coagulopathy** should be corrected before surgery.
- Genitourinary**
Urinary tract infection.
- Neurological:** Epilepsy.
- Endocrine/metabolic**
Diabetes mellitus (should be properly controlled before elective surgeries).
- Infectious diseases:** HIV, hepatitis and TB.
- Previous surgery:** Type of anesthetic and any problems encountered (either with the patient or one of his relatives).
- Social history**
Patient's occupation.
- Drug history:** Illegal drug abuse.

Physical examination

This will include:

- | | |
|------------------------|---------------------|
| A. General examination | D. Gastrointestinal |
| B. Cardiovascular | E. Genitourinary |
| C. Respiratory system | F. Neurological |

Investigations

(Apart from routine investigations, others are done only when clinically indicated)

- Routine investigations**
 1. Urine analysis, CBC, coagulation profile (BT, PT, PTT).
 2. ABGs, pH and electrolytes (Na^+ , K^+ , Cl^-) and Fasting blood sugar (FBS).
 3. Kidney function tests (KFTs), Liver function tests (LFTs).
- Cardiovascular:**
ECG, echo, ejection fraction.

C. Respiratory:

Chest X-Ray, screening for lung tumors, FEV₁.

D. Endocrine

Pre-operative Preparation

1- **Consent:** must be signed before the operation.

2- **Preoperative round:**

- Final re-examination of the patient.
- Marking the side to be operated upon (in cases of unilateral conditions).
- Making sure that investigations are completed.
- Preparation of blood if transfusion is needed.

3- **Diet:**

- High caloric diet should be taken on the day before the operation.
- **Special preparation in large bowel surgery** → low residue diet.
- The patient should be fasting at least 6-8 hours before operation.

4- **Bowel preparation:**

- Purgatives are no more advisable.
- Soap and water enema may be given in rectal and major operations.
- **In GIT surgeries** → NGT is inserted (better after induction of anesthesia).

5- **Skin preparation:**

- On the morning of operation, shower with 10% povidine-iodine.
- The skin of the operation is shaved, scrubbed with soap and water and antiseptic, and then covered with a sterile dressing.

6- **Maintenance of fluid balance:**

- IV infusion is started in theatre.
- Urinary catheter may be needed to monitor urine output (inserted after the patient is anesthetized).

7- **Pre-anesthetic medications:**

- ⇒ **Timing:** usually given 0.5-2 hours before the induction of anesthesia.
- ⇒ **Selection is largely subjective:** not every patient needs all these medications.
- ⇒ **Goals:**

1. Sedation/anxiolysis.
2. Analgesia.
3. Reduce airway secretions/heart rate control.
4. Prevent bronchospasm.
5. Hemodynamic stability.
6. Prevent and/or minimize the impact of aspiration.
7. Decrease post-op nausea/vomiting.
8. Antibiotic prophylaxis.

Sedation

- By **benzodiazepines or opioids (or both)**

Analgesia

- I.M. Morphine 0.05 - 0.15 mg/kg.
- **Side effects:**
 - Nausea
 - Decrease gastric motility,
 - Biliary spasm
 - Respiratory depression especially with benzodiazepines

Airway secretions/Heart rate & prevention of bronchospasm

- **Anticholinergics:** for reduction of secretions.
- **Glycopyrrolate:** β_2 agonist for bronchospasm.

Hemodynamic stability

- Anti-hypertensives.
- For urgent or emergent surgery in patients with poorly controlled hypertension Labetalol is used.

Decrease gastric acidity

- H2 antagonists, PPIs.

Nausea / Vomiting

- Ondansetron.

Medications to be continued

- **Cardiovascular medication:**
calcium-channel blockers, beta blockers, nitrates, and anti-arrhythmic.
- **Bronchodilator**
Should be continued, as withdrawal can precipitate bronchospasm.
- **Antiepileptic treatment:**
- **Steroid intake:**
Sudden withdrawal causes Addisonian crisis
- **Aspirin**

Medications to be stopped

- **Anticoagulants**
 - Warfarin should be stopped at least 4 days before surgery
 - In patients requiring DVT prophylaxis, spinal or epidural can be performed 6 hours after subcutaneous injection of unfractionated heparin and 12 hours after low molecular weight heparin.
- **Contraceptive pill and anesthesia. Synthetic estrogens**
- **Monoamine oxidase inhibitors**
- **Selective serotonin reuptake inhibitors (SSRIs).**

Surgery in High Risk Patients

- ➡ See aid to post-graduate book

Surgery in Diabetes Mellitus

- ➡ See aid to post-graduate book

Some Anesthetic Considerations for Patients with Medical Problems

- ➡ See aid to post-graduate book

Anesthesia

Definition

- It is the controlled and reversible depression of the normal response of the whole or part of the body by chemical agents called anesthetics.

Types of Anesthesia

I. General

II. Local (Regional)

A. Central neuro-axial block

- Spinal
- Epidural

B. Peripheral sensory nerve block

- Surface (topical) analgesia
- Local infiltration
- Nerve block
- Local intravenous analgesia

I. General Anesthesia

Definition

- Controlled and reversible depression of the functional activity of the CNS.

Triad of general anesthesia

- Unconsciousness (hypnosis).
- Analgesia.
- Muscle relaxation.

Anesthesia Tools (Drugs & tools)

A. Categories of anesthesia drugs:

- Intravascular induction agents:** to provide rapid smooth induction of hypnosis e.g. barbiturates – non barbiturates.
- Gases and volatile liquids:** given by inhalation to diffuse rapidly from the lungs to the brain e.g. nitrous oxide, halothane, isoflurane.
- Analgesics:** include opioid (as morphine or its synthetic derivatives as pethidine) and non-opioid analgesics as NSAIDs.
- Muscle relaxants and their antagonists:** muscle relaxants act at the neuromuscular junction (NMJ) by blocking the binding of acetylcholine to nicotinic acetylcholine receptors.

B. Anesthesia equipment:

- Airway equipment:** patency of airway should be ensured by oral airways, endotracheal tubes or laryngeal mask.
- Anesthesia machine:** the main components of anesthesia machine are a flowmeter to adjust flow of gases and vaporizers for administration of volatile anesthetics.
- Anesthesia ventilator.**

4. Monitoring during anesthesia:**a. Vital data:**

- Pulse (ECG) and blood pressure.
- Temperature.
- Respiratory monitoring (oxygen saturation).

b. Neuromuscular monitoring: by a device that stimulates a peripheral nerve and records the response of the muscles supplied by it.

Phases of general anesthesia**A. Induction:**

- Intravenous is the most commonly used.
- Recently → inhalational, intramuscular and **rectal**.

It includes:

- A. Induction.
- B. Maintenance.
- C. Recovery.

B. Maintenance:

1. Inhalational anesthetics (recently → intravenous maintenance can be used = total intravenous anesthesia, TIVA e.g. if surgery on airway).
2. Muscle relaxants.
3. Additional doses of narcotics.
4. Mechanical ventilation of paralyzed patients.
5. Close monitoring of vital data.

C. Recovery:

1. Discontinuation of inhalational anesthetics.
2. Antagonism of muscle relaxants by neostigmine with atropine.
3. Discontinuation of mechanical ventilation.
4. Oro-pharyngeal suction and removal of endotracheal tube.
5. Transfer the patient to the **post anesthesia care unit (PACU)** or surgical intensive care unit (SICU).

Complications of general anesthesia**A. Complications during induction of anesthesia:**

1. **Airway manipulation:** e.g. failure to intubate – undetected esophageal intubation – laryngospasm – reflex bronchospasm aspiration of gastric content.
2. **Stress response to intubation:** e.g. hypertension – tachycardia – arrhythmias.
3. **Administration of intravenous drug:** e.g. allergic drug reaction – histamine release leading to bronchospasm and hypotension – injection of wrong drug – awareness during anesthesia – drug overdose leading to hypoventilation and hypotension.

B. Complications during maintenance of anesthesia:

1. Inadequate fluid or blood replacement.
2. Nerve injury due to inappropriate patient position.
3. Undetected patient ventilator disconnection.
4. Corneal ulcer due lack of protective reflexes.
5. Malignant hyperthermia which is a rare but very serious complication.

C. Complications during recovery from anesthesia:

1. Post-operative nausea and vomiting.
2. Sore throat from endotracheal tube replacement.
3. Inadequate recovery of muscle power.

D. Delayed complications of anesthesia:

1. Muscle pain due to fasciculations caused by succinyl choline.
2. Halothane induced hepatotoxicity.

How to minimize complications associated with anesthesia?

1. Proper pre – operative evaluation and preparation.
2. Before induction of anesthesia, the anesthetist must:
 - Check the equipment to be used.
 - Prepare all drugs including emergency drugs.
3. Proper monitoring of the patient intra-operatively.
4. Call for help early when needed.

II. Central Regional Anesthesia

	Spinal	Epidural
Site of injection	Subarachnoid directly into the CSF (intra-theal)	Between dura matter and periosteal lining of vertebral canal
Onset	Rapid (within 5 min.)	Slow
Duration of action	Short acting	Adjustable (according to the dose)
Advantages	1. Rapid onset of action 2. Adequate analgesia	1. Slow sympathetic block → controlled hypotension → less blood loss 2. Multiple shots can be given → adjust the effect and duration 3. Can be given at various levels of the vertebral column
Disadvantages & complications	1. <u>Rapid sympathetic block</u>: • Severe hypotension & bradycardia (→ add IV fluids ± vasopressors) • Ventilator failure (→ ventilator) → to minimize → inject below T10 2. <u>Single shot</u> (can't be repeated → short acting → add epinephrine to prolong 3. <u>Post-Dural-Puncture Headache (PDPH)</u> → now minimized by using special needles	1. <u>Urinary retention</u> → use catheter ± prophylactic α-blocker 2. <u>Inadequate analgesia (if used alone)</u> → add opioid analgesics N.B. opioid analgesic → respiratory depression in the 1st 24 hours (→ monitoring)

Indications

- 1) Operations below the umbilicus as inguinal hernia, anal operations, operations on the urinary bladder or lower limb amputations.
- 2) In patients with respiratory tract infection.
- 3) In presence of renal or hepatic problems.
- 4) In emergency situations when the patient has a full stomach.

Advantages

- 1) It is simple and easy to be performed.
- 2) It gives adequate muscular relaxation.
- 3) It has minimal interference with cardiac, hepatic or renal functions.

Contraindications

A. Absolute

- 1) Patient refusal.
- 2) Known allergy to local anesthetic drugs.
- 3) Infection or local sepsis at the site of needle insertion (lumbar region).
- 4) Bleeding or coagulation disorders (or patients receiving anticoagulants).

B. Relative

- 1) Hypovolemia.
- 2) Low fixed cardiac output.
- 3) Systemic sepsis.
- 4) Deformities of the spine as kyphosis.
- 5) Neurological or psychological disturbances for medico-legal causes.

III. Local Regional Analgesia

Types

1. Surface analgesia:
 - By local anesthetic spray, or cream.
 - It is used for mucous membranes as the mouth.
2. Local infiltration analgesia.
 - By local injection of the anesthetic in the subcutaneous tissue to block the sensation in a localized area
3. Nerve block.
 - Local injection of the anesthetic in the neural sheath around a peripheral nerve.
4. Local intravenous analgesia.
 - It is used in the extremities after application of a tourniquet.

Indications

- Minor surgical procedures.
- When general anesthesia is contraindicated e.g. in patients with compromised cardio-respiratory function.

Advantages

- Effects of the drug are limited to the part of body to be operated upon.

Relative contraindications

- In children under 10 years.
- In psychotics.
- Non-cooperative patients.

Post-operative Pain Management

Factors affecting response to analgesia

- Site of operation.
- Age and sex.
- Psychological factors.
- Anesthetic management.
- Bodyweight.

Non-opioid agents

- Used for mild to moderate pain
- Drugs
 - Paracetamol
 - NSAID's
 - Ketorolac IV/IM
 - Diclofenac PO/IM/PR
 - Compound analgesics: paracetamol/codeine

Opioid agents

- Properties:
 - Analgesia.
 - Cough suppression.
 - Histamine release (avoid in asthmatics).
 - Nausea & vomiting.
 - Biliary spasm (avoid in biliary colic).
 - Tolerance.
 - Ventilatory depression.
 - Vasodilatation.
 - Constipation.
 - Pupillary constriction (avoid in head injury).
 - Urine retention (avoid in renal colic).
 - Dependence.
- Drugs:
 - Morphine.
 - Pethidine.
 - Tramadol.
 - Fentanyl.
- Types of administration:
 - Conventional intermittent - morphine 10 mg IM 2-4 hourly.
 - Parenteral routes: - Bolus. - Continuous infusion - rate 3-5 mg/hr.
 - Non-parenteral routes:
 - Sublingual - buprenorphine.
 - Oral.
 - Rectal.
 - Transdermal - fentanyl.

Pain control using local anesthetics

- Local infiltration of site.
- Spinal.
- Epidural.
- Extradural.
- Regional blockage.

Other methods

- Transcutaneous electrical stimulation.
- Cryotherapy.
- Acupuncture.

About morphine

- 1- Avoid it in biliary operations.
- 2- Avoid it in coma, CNS problems, respiratory depression.
- 3- Shouldn't be repeated → addiction.
- 4- Withdrawal side effects:
 - Agitation.
 - Vomiting.
 - Diarrhea.
 - Agrenion.

Post-Operation

1- Postoperative Care

➡ See aid post-graduate book.

2- Postoperative Complications

GENERAL COMPLICATIONS:

- **Cardiac complications:** hypertension, arrhythmias, MI & heart failure.
- **Respiratory complications:** postoperative atelectasis, bronchitis, bronchopneumonia, ARDS.
- **DVT & pulmonary embolism.**
- Nausea & vomiting.
- **Thrombophlebitis of a peripheral on central venous line.**
- **Fever:** may be due to:
 - Reaction to surgery: may occur in the 1st 24hrs.
 - Pulmonary complications (usually the cause in the 1st few days).
 - Surgical site infection: usually in the 4th or 5th day, but it may be delayed.
 - Thrombophlebitis of a peripheral or a central venous line.
 - DVT & pulmonary embolism.
 - UTI.
 - After abdominal surgery (leakage of an anastomosis or development of intra-abdominal abscess).
- **Neurological complications:** stroke or transient ischemic attacks.

LOCAL COMPLICATIONS:

- Seroma.
- Hematoma.
- Wound dehiscence.
- Wound infection.
- Hypertrophic scar & keloid.

Complications may follow abdominal surgery:

- Acute gastric dilatation.
- Paralytic ileus
- Adhesive intestinal obstruction.
- Leakage of an intestinal or gastric anastomosis → may lead to peritonitis, intra-abdominal abscess or fecal fistula.
- Intra-abdominal abscess (pelvic, interloop or subphrenic).
- All the previous complications will lead to nausea & vomiting.

Day Case Surgery

➡ See aid post-graduate book.

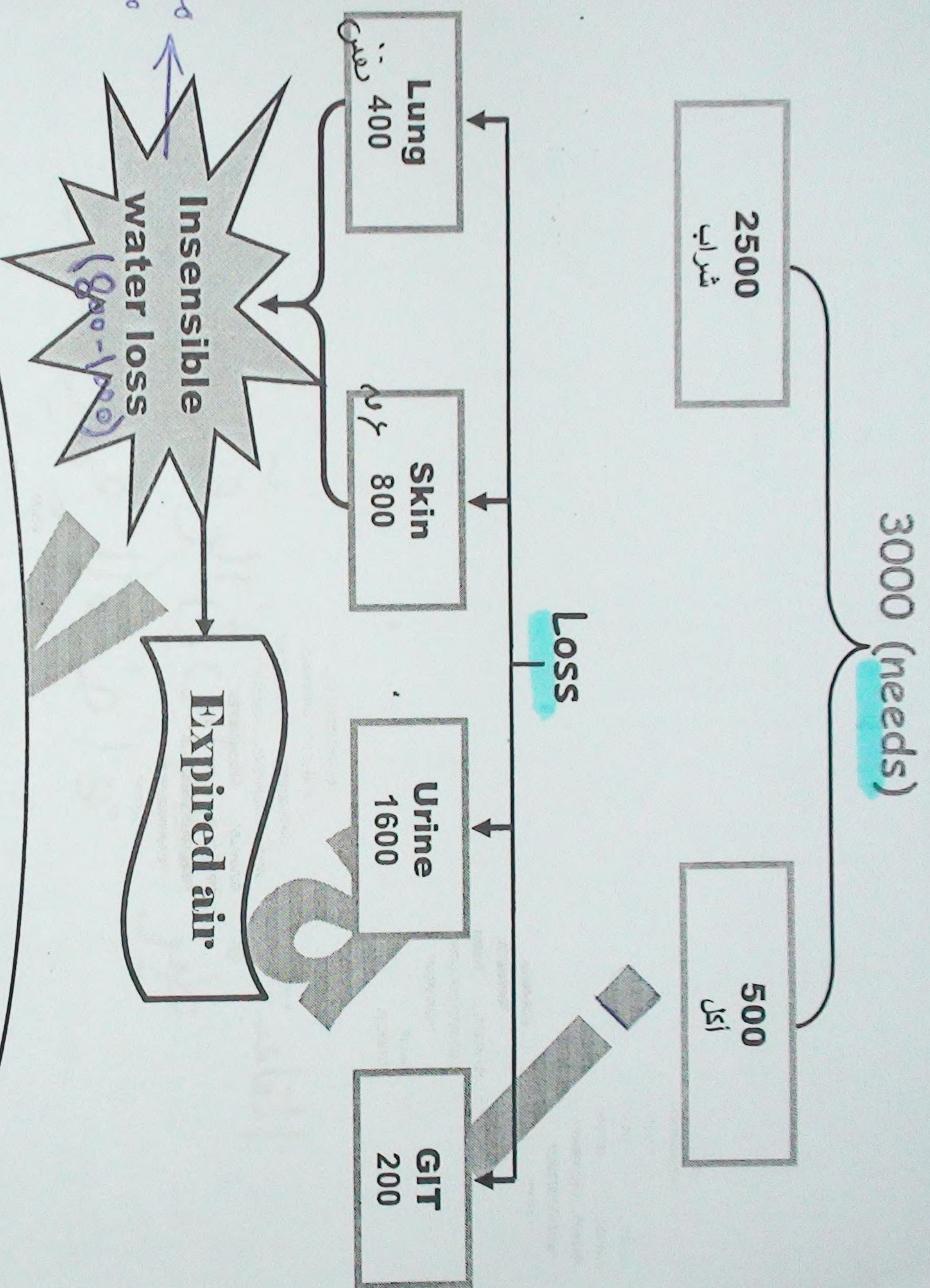
Water & Electrolyte
imbalance

PC

Dr. Mohamed El_Matary



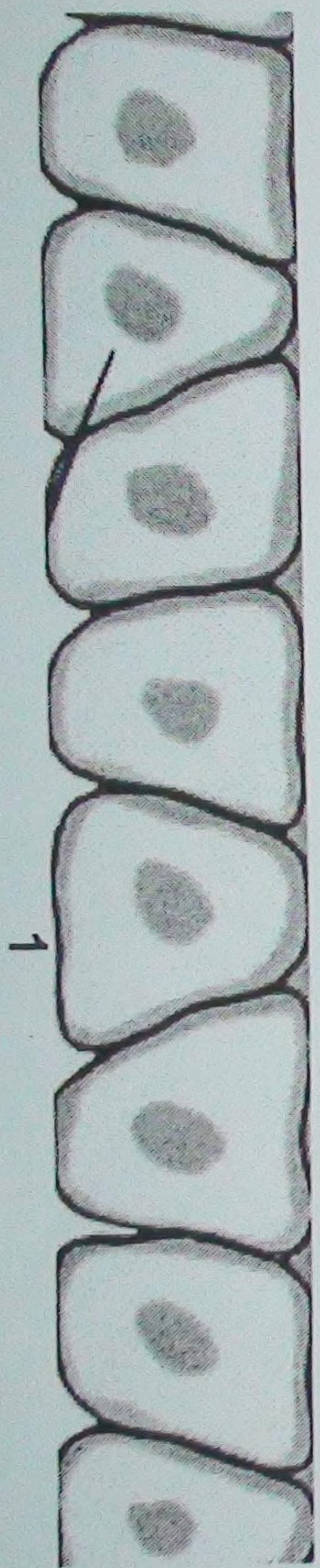
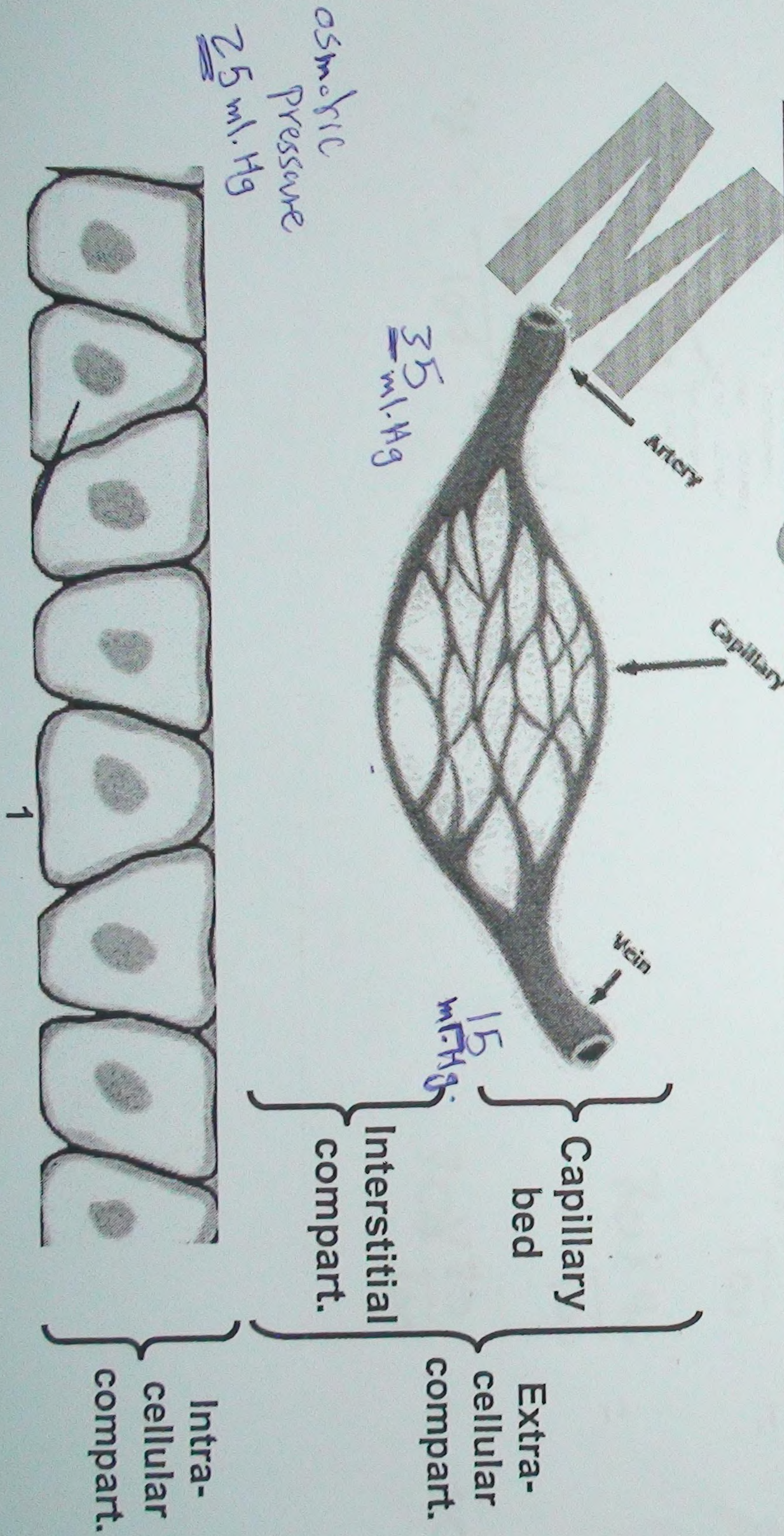
Body water (ml.)



H₂O represent 60% of body constituents
 H₂O more in cells > 75%

العيب في حدود 100 واختار

Water in the body compartment



القاعدة:
 غير المثل في الرصيد
 و: مائة الرصيد

$$\begin{array}{r} \frac{60}{100} \times 70 = 42 \\ 60 \times 70 = 4200 \end{array}$$

$$70 \times \frac{60}{100} =$$

$$70 \times \frac{40}{100} =$$

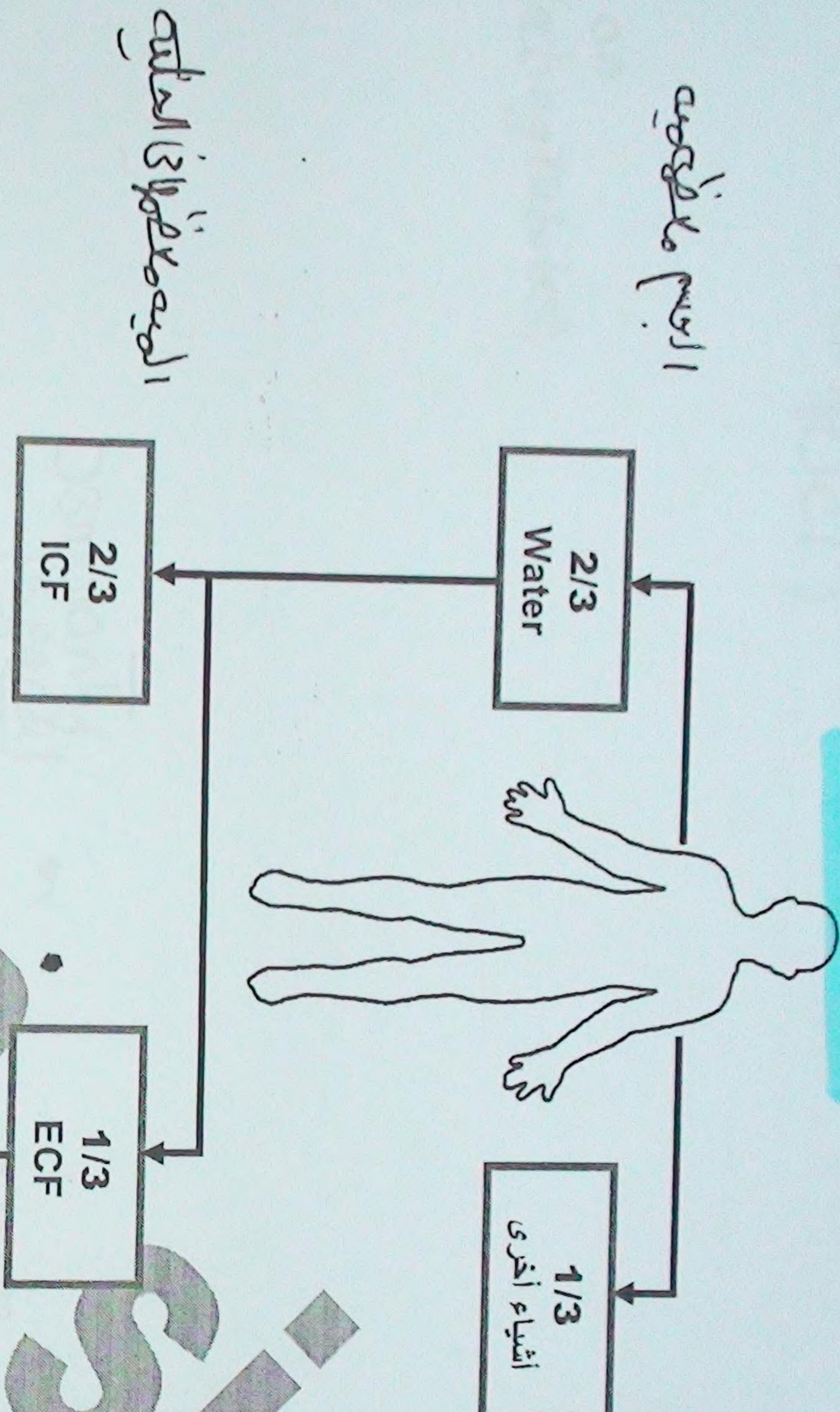
$$70 \times \frac{20}{100} = ECF$$

الماء في الجسم

H₂O content is more in muscular

العضلات تحتوي على الماء أكثر من بقية الأنسجة

قاعدة ال 2/3



ملاحظات

Body water by %

Examples:

- Patient weight is 70 kg → so, 10% = 7
- Patient weight is 60 kg → so, 10% = 6

Body 70 kg



40%
أشياء أخرى

60%

42 L =

40%
ICF

28 L =

14 L =

20%
ECF

14%
ISF

10 L =

4 L =

6%
Blood (plasma)

يعني بحال الله
أقل نسبة في الجسم
موجود في الدم

لوحد وزنه 70 كيلو

← على الماء 7%

وله 60% من الماء، 10% من الماء

الماء في الجسم
الماء في الجسم
الماء في الجسم

* ADH depends on osmolality
 * Aldosterone depends on Na⁺ & K⁺ levels
 on DCT

act on
 (collecting tubules)

Osmolality
 is equal
 in both

ECF $\leftrightarrow$ ICF

300

300

↳ ions
 Na⁺, Cl⁻
 HCO₃⁻

↳ ions
 K⁺, Mg²⁺, Ca²⁺, org. P.

(pressure > 60 mmHg)

سيكون 111 <

Isotonic 0.9 NaCl

expand ECF - not affect intracellular

hypotonic Saline $\rightarrow$ expand intracellular only

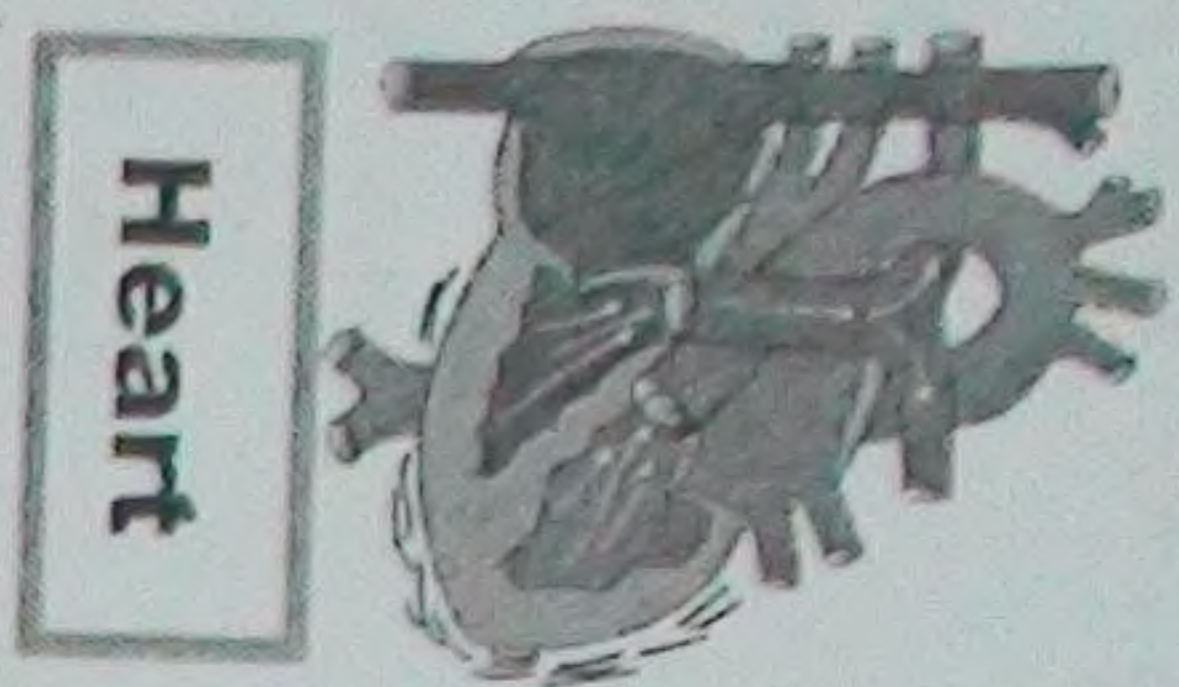
سيكون 120 < 111

↓
 (pressure 1500)

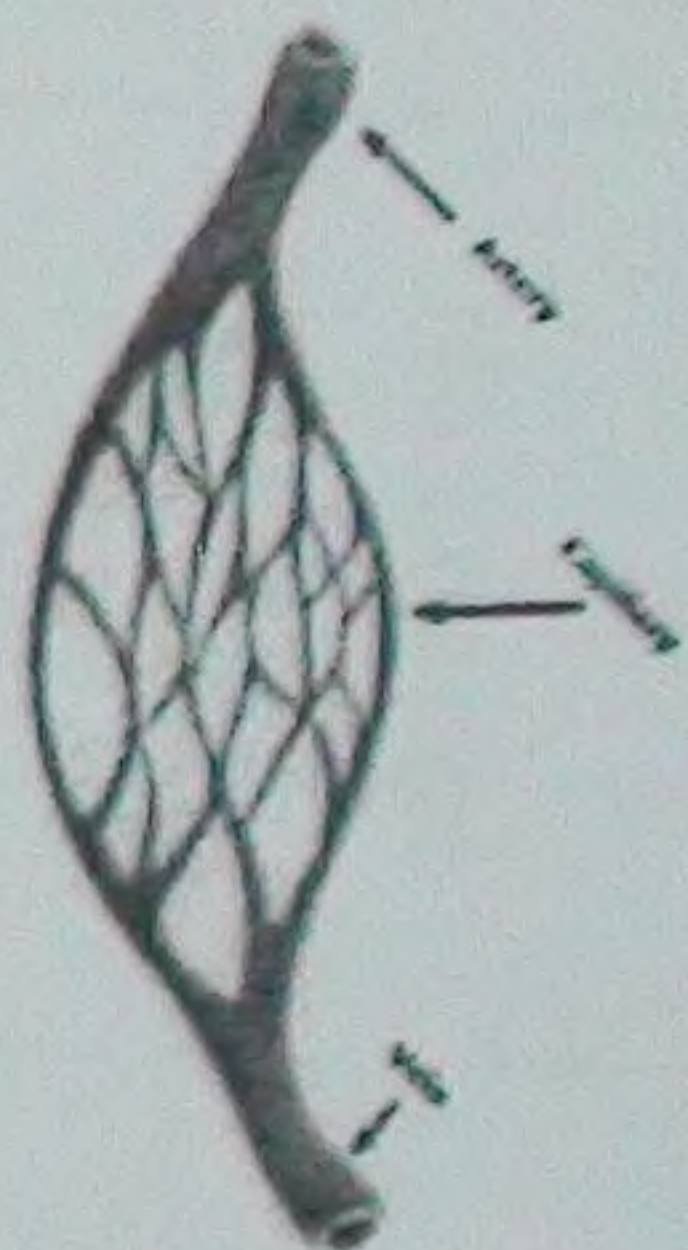
hypertonic Saline

$\rightarrow$ expand ECF
 & absorb H₂O from intracellular

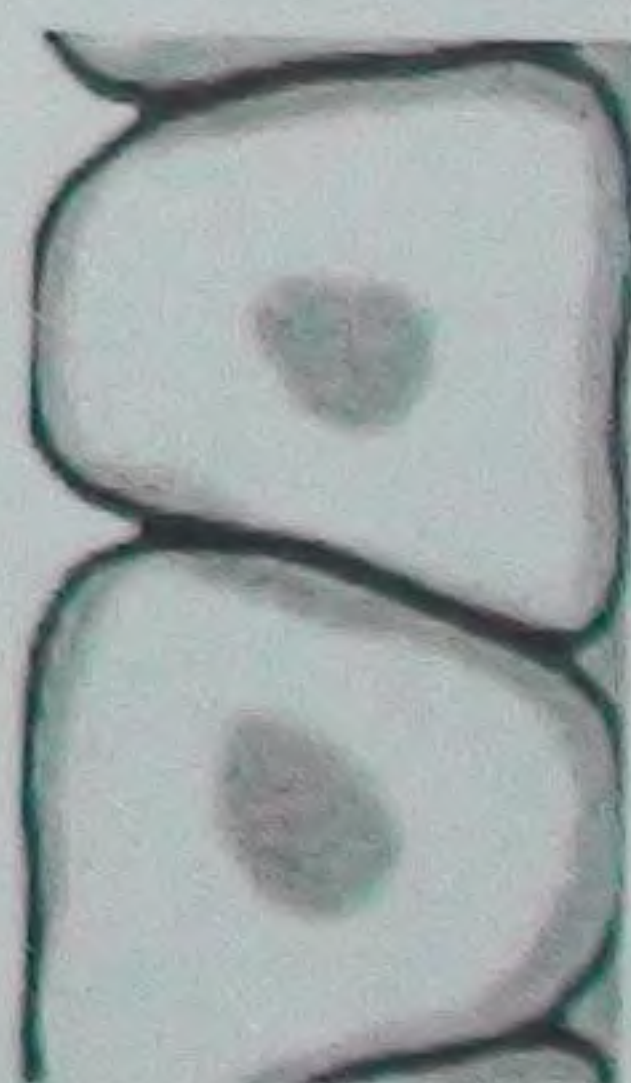
حركة الماء (Normal)



Heart



Hydrostatic pr. (oncotic pr.)



Na⁺-K⁺ pump

صورة الوقت
التي cell يتم
بها Na⁺ و K⁺
ويرذل ATP

ملحوظة:
Osmolarity of the plasma protein، حركة الماء، هناك اثنان كامل في لتعويض ال Osmolarity of the plasma protein، حركة الماء، هناك اثنان كامل في لتعويض ال

152 meq/L

▶ Osmolarity of the body = 270 - 290 mOsmol (تقريباً double the Na⁺ level)

▶ Osmolarity of ECF → depends on → [Na⁺ + Cl⁻ + HCO₃⁻] 22 - 28 meq/L

▶ Osmolarity of ICF → depends on → [K⁺ + organic phosphorus]

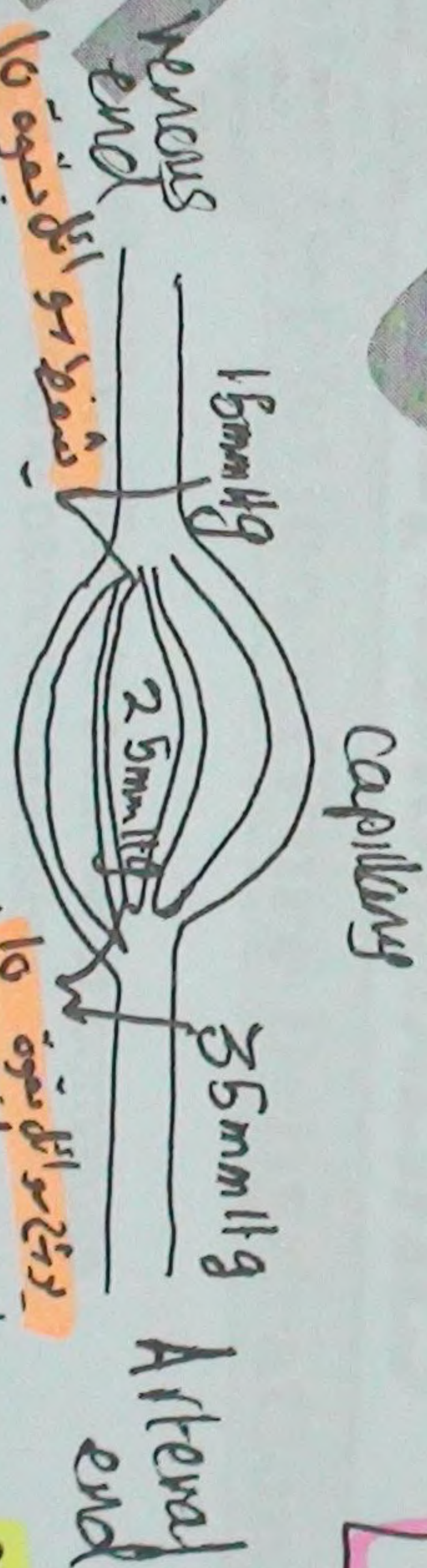
▶ ADH is 1ry responsible for control of plasma osmolarity & is most sensitive to plasma Na⁺ concentration

▶ The volume of ICF doesn't depend on or respond to volume of ECF, ICF changes only when ECF osmolarity changes

[↓ ECF osmolarity → expansion of ICF volume]

[↑ ECF osmolarity changes → ↓ volume of ICF]

Na ⁺	135-145 meq/L
Cl ⁻	102
HCO ₃ ⁻	22-28
K ⁺	153



* osmotic pressure depends only on proteins

« لا يتأثر الضغط الأسموزي بوجود الأيونات في الدم، فقط بروتينات الدم »

* Mainly → Albumin is the main contributor

* Albumin = 3.5 - 5 gm/dL

* If ↓ → NO oncotic pr. → edema

قوة الضغط قلت

lymphedema

Same mech. as Cardiac edema

Non pitting but lymphedema can remain for years - Exchange of part of it → Non pitting

► **This table shows:**

- Volume changes + body osmolarity fall changes in body hydration

	ECF volume	Body osmolarity	ICF volume	
Loss of isotonic fluid e.g. - Hge - Diarrhea - Vomiting	↓	No change	No change	Hypovolemic iso-osmotic
Loss of hypertonic fluid e.g. - Aldosterone deficiency (Addison ds.)	↓	↓	↑	Hypovolemic hyposmotic (loss of solutes > loss of water)
Loss of hypotonic fluid e.g. - Dehydration - Diabetes insipidus - Alcoholism (suppress ADH)	↓	↑	↓	Hypovolemic hyperosmotic (loss of water > loss of solutes)
Gain of isotonic fluid Isotonic saline infusion	↑	No change	No change	Hypervolemic iso-osmotic
Gain of hypotonic fluid - Hypotonic saline - Water intoxication - SIADH	↑	↓	↑	Hypervolemic hypotonic
Gain of hypertonic fluid: - Hypertonic saline infusion - Hypertonic mannitol infusion - Mineralocorticoid excess (1ry or 2ry hyperaldosteronism)	↑	↑	↓	Hypervolemic hyperosmotic

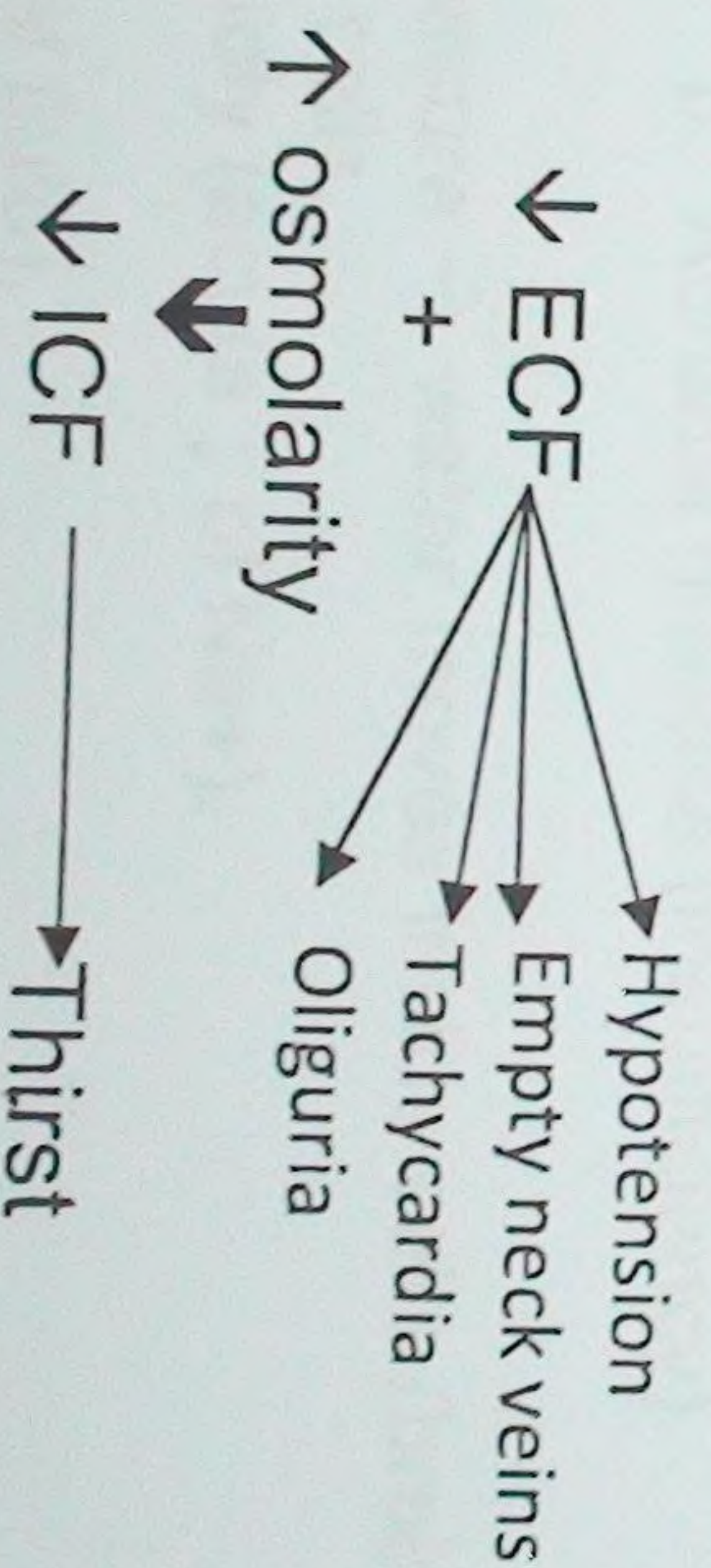
WATER ABNORMALITIES

Water Depletion = (Pure Dehydration)

Causes

- ↓ intake: ↓ availability, difficult to swallow or comatose.
- ↑ output: as in fever and osmotic diuresis.

Clinical Picture



Management

- Replace by **sodium free water**
- If severe depletion, at least 1/2 the estimated deficit should be replaced in the initial 12 hours,
- IV glucose 5%** the purpose is that glucose makes the solution isotonic to (ECF).

Water overdose = (Water toxicity)

Causes

Increased intake:

1. Pre operative water enema.
2. Operative: trans-urethral resection of prostate (T.U.R.P) syndrome.
3. Post operative over infusion of 5% glucose IV (commonest).
4. Disease: Neurosis (excessive compulsive intake).

hysterical

Pathophysiology

- Dilutional Hyponatremia

Clinical Picture

- ECF $\rightarrow$ $\downarrow$ osmolarity & $\uparrow$ volume $\rightarrow$ Hypertension & congested neck veins
Shift fluid to ICF $\rightarrow$ edema of cells $\rightarrow$ (brain) edema, convulsions, $\uparrow$ ICT & vomiting

Management

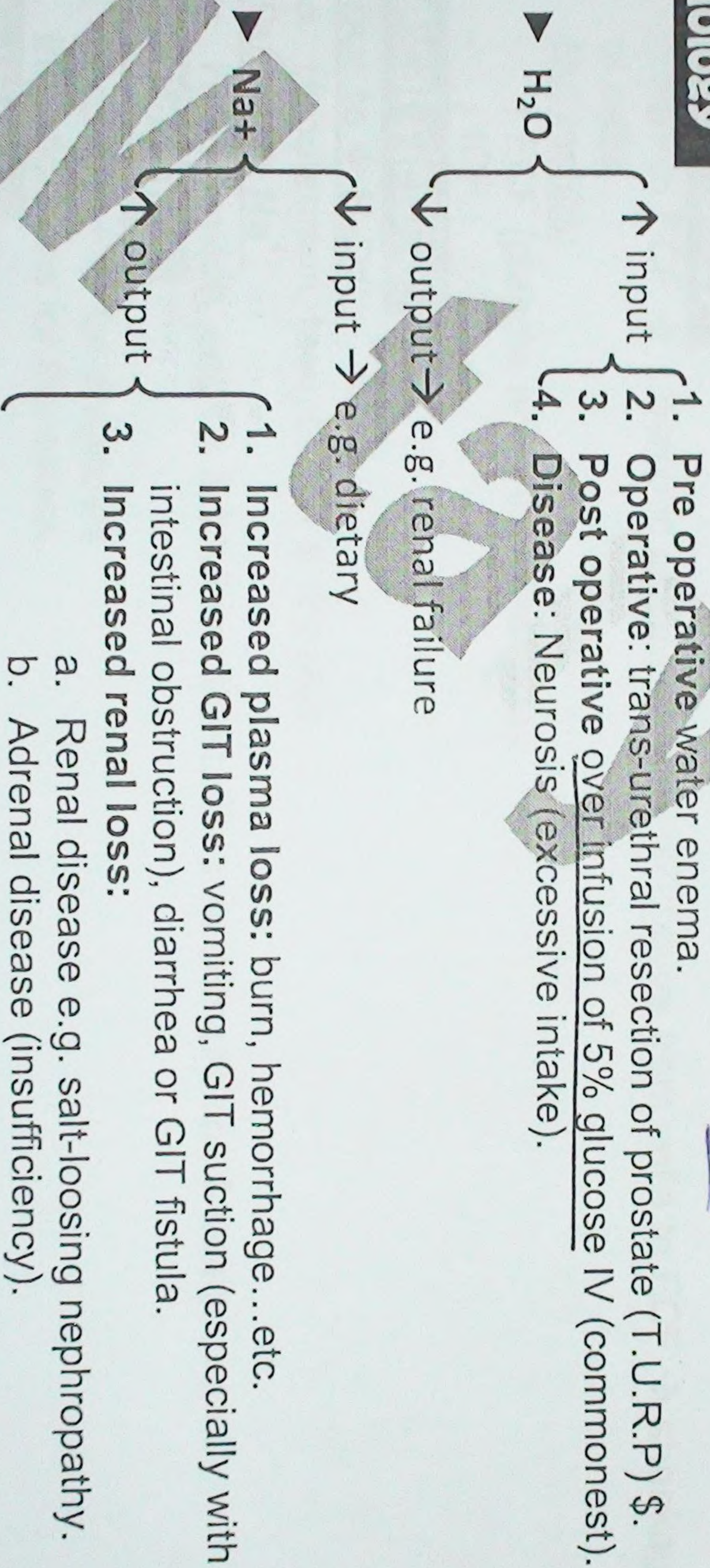
1. Mild $\downarrow$ water intake $\rightarrow$ water restriction
2. If renal failure $\rightarrow$ dialysis.
3. If brain edema $\rightarrow$ hypertonic saline. $\rightarrow$ if $Na^+ < 120$

HYPONATREMIA = Shock $\rightarrow$ hypotension

Definition

- Decreased serum $Na^+ < 130$ mEq/L.

Etiology



Pathophysiology

- $\downarrow Na^+$ in ECF $\rightarrow$ $\downarrow$ osmotic pressure $\rightarrow$ water moves inside cells (brain cells are the most dangerous) $\rightarrow$ swelling of cells.
- This is more severe if occurs rapidly (acute $\downarrow$ of Na^+).
- Net results:
 - $\downarrow$ ECF (plasma and interstitial fluid).
 - $\uparrow$ ICF (especially if acute reduction).

Clinical picture

1. Due to $\uparrow H_2O \rightarrow$ as water intoxication.
2. Due to $\downarrow Na^+ \rightarrow$ as shock $\rightarrow$ mainly signs

1. cl/p.p. of cause
2. Tachycardia & weak pulse
3. hypotension
4. sub normal Temp.
5. cold skin

Investigations

- Serum Na^+ $\rightarrow$ for diagnosis.
- Investigations for the cause e.g. renal failure.

Treatment

A. Active treatment:

- Mild to moderate cases $\rightarrow$ normal saline infusion (0.9% NaCl).
- Severe cases $\rightarrow$ 5% NaCl.

B. Treatment of the cause.

Prognosis

- Mortality rate ranging from 5 – 50% according to severity of the case

HYPERNATREMIA

Definition

- Increased serum ($\text{Na} > 145 \text{ mEq/L}$)

= HTN or edema or $\swarrow$
water depletion

Etiology

A. Due to $\downarrow \text{H}_2\text{O}$:

- Loss of water as osmotic diuretics
- Inadequate water intake as old age

B. Due to $\uparrow \text{Na}^+$:

- Post-operative (excessive saline administration)
- Conn's syndrome
- Cushing's syndrome.

Pathophysiology

- $\uparrow \text{Na}^+$ in ECF $\rightarrow$ $\uparrow$ osmotic pressure $\rightarrow$ water moves from cells to ECF $\rightarrow$ shrinkage of cells.
- Net results:
 - $\uparrow$ ECF (plasma and interstitial fluid).
 - $\downarrow$ ICF.

Clinical picture

1. Due to $\downarrow \text{H}_2\text{O}$:

- Hypotension, tachycardia & dry skin.

2. Due to $\uparrow \text{Na}^+$:

- Hypertension, edema & CNS manifestations as convulsions & coma.

Investigations

- Serum Na^+ $\rightarrow$ for diagnosis.
- Investigations for the cause.

Treatment

- A. Active treatment: **sodium-free water** (to be correlated with the duration of hypernatremia).

if severe $\rightarrow$ glucose 5%.

B. Treatment of the cause.

Prognosis

- Most patients survive, but with residual neurological deficit that becomes persistent in up to 30%.

Potassium

- ▶ The percentage of potassium in the ECF is 2%.
- ▶ Daily needs = 60 mOsmol/L.
- ▶ Normal level:

MCQ

- In ECF = 5 – 5.5 mmol/L.
- In ICF = 150 mmol/L.

Physiology

- Most of the K is **intracellular 98%** & stored mainly in **skeletal muscles 2%** ms - org ms
- Intake:
 1. 90% of oral K is absorbed and buffered almost immediately intracellularly by the increased **insulin** secretion and the basal catecholamine level.
- K transfer into cells:
 1. The K is kept intracellular against concentration gradient by **Na/K ATPase pump**.
- Factors increasing K transfer **inside the cells** is: **insulin**, **B2 stimulation**, **alkalosis**
- K movement **outside** the cells is facilitated by: **acidosis**, **hyperosmolarity** and **B-blockers**
- K excretion:
 1. 90% of filtered K is **reabsorbed** in the **proximal convoluted tubules** and **loop of Henle**.
 2. K is **then re-secreted** in the **lumen of distal convoluted tubule** due to Na reabsorption that creates **intraluminal negative pressure**. This excretion is enhanced by **aldosterone**, increased Na delivery to DCT, and hyperkalemia. Since H⁺ competes with K for excretion, alkalosis favors K excretion.
 3. 10% of K is excreted through the **GIT**.
 4. **Highest potassium concentration occurs in succus entericus.** سكس انتريكس

Definition

- Serum K < 3.5 mEq / L

Etiology

1. Decreased intake:
 - IV fluid therapy deficient of potassium.
 - Prolonged decrease of dietary intake
2. Decreased absorption: malabsorption syndrome
3. Increased loss:
 - A. Renal loss:
 - Diuresis:
 - Diuretics especially loop diuretics & thiazides
 - diuretic phase of acute tubular necrosis
 - Osmotic diuresis e.g. fanconi syndrome, RTA
 - Conn's syndrome & Cushing's syndrome (hyperaldosteronism).
 - Alkalosis
 - Drugs: e.g. diuretics.
 - Antibiotics e.g. carbenicillin

Hypokalaemia

2.5

4.5

→

واعتبر K^+ باء

عازي اوس

$$4.5 - 2.5 = 2 \text{ mme}$$
$$\times 100 = \boxed{200 \text{ mL}}$$

عازي اقسيم كال

عزل في باء

بقي محتاج

5 باءات

B. Gastrointestinal loss:

1. Diarrhea especially secretory diarrhea (cholera & ulcerative colitis) → the commonest cause
2. Vomiting.
3. Gastric suction and external GIT fistula.
4. T. tube.
5. Intestinal fistula (e.g. in chron's disease)
6. K⁺ losing tumor (villous adenoma). carpet tumor

A. excessive tapping of ascites

4. Intracellular shift:

- o Alkalosis
- o Insulin therapy
- o Hypokalaemic periodic paralysis.

Hazards

- 1- ↓↓ nerve and muscle excitability.
- 2- ↑↑ risk of supraventricular arrhythmias in patients taking digoxin.
- 3- ↑↑ risk of hepatic coma in patients with liver disease.
- 4- Polyurea due to ↓ responsiveness to antidiuretic hormone.

Clinical Picture

- Mild case: asymptomatic

I- Clinical picture of the cause.

II- Effect of hypokalemia on:

- Smooth muscles → paralytic ileus.
- Skeletal muscles → hypotonia & hyporeflexia. (ASThenia)
- Cardiac muscle → Arrhythmias especially in digitalized patients & cardiac arrest.
- Brain → (apathy)
- Kidney → renal tubular damage (hypokalaemic nephropathy) (nephrogenic diabetes insipidus).
- Liver → liver damage especially in patient with liver disease.

Investigations

1- Serum K:

- Is reduced (normal: 3.5-5 mEq/L).

2- ECG:

- Sagging depression of ST segment.
- Flat or inverted T wave
- Prominent U wave

3- Investigations for the cause.

Treatment

1. Treatment of the cause.

2. Potassium replacement:

- Oral potassium (in mild cases)
- IV potassium should be given slowly in a large vein with continuous ECG monitoring in ICU.
- Estimation of potassium deficient = (4.5 - serum K concentration) X 100 in normal pH in adult.
- Safe rules for giving K are:
 - o Urine output at least 40 ml/hour.
 - o No more than 40 mmol added to 1 L fluid.
 - o No faster infusion than 40 mmol/Hour. ≠ Not more than 1 L/hour

TTT OF ↓↓ K RED 100

- Rule of 40
- Estimate
- Decide oral or IV



Acute Tubular Necrosis → oliguric phase
 → Reverse
 إذا قلنا، فإنه إذا كان البول قليلاً، فذلك
 hypernatremia هو

Then
 Cardiac necrosis → oliguric phase
 hypernatremia هو

* in Shock → urine out put is monitored
 indicator of kidney function
 & other organ perfusion

if M^+ reaches
5.5 - 6.5 → Supravent. Tachycardia
 †† → glucose & insulin
 if Reaches (7) → dialysis
 chelating Agent (EDTA)

Hyperkalaemia

Chronic
Renal failure

Definition

Serum K > 5.3 mEq / L

Etiology

1- Increased intake:

- Excessive therapy with IV potassium (the commonest cause).
- Transfusion of stored blood

2- Decreased Excretion:

A. Renal cause: (the 2nd common cause)

- Acute renal failure
- Severe chronic renal failure
- Tubular disorder e.g type IV RTA
- B- Adrenal cause: Addison's disease hyperpigmentation - hypotension - hypoglycaemia
- C- Drugs: Potassium-Sparing diuretics

3- Extracellular shift:

- Tissue damage:
- Haemolysis
- Rhabdomyolysis
- Tumour lysis syndrome as in lymphoma pt After chemotherapy
- Acidosis
- Insulin deficiency
- Hyperkalaemic periodic paralysis
- Drugs e.g Succinylcholine.

4- Pseudohyperkalaemia:

- Polycythaemia
- In vitro haemolysis

Clinical Picture

- Apathy.
- Arrhythmia ~~not~~ cause of death

Diarrhea
Colic.

≠ ↓ K⁺
↳ paralytic illness

Investigations

1. Serum K: is increased (normal 3.5-5 mEq/l).

2. ECG:

- Prolonged P-R interval
- Loss of P wave
- Widened QRS complex
- Bradycardia, heart block, sine wave pattern & arrest

3. Investigation for the cause

Treatment

1. Treatment of the cause.

2. Correction of hyperkalaemia:

- If K > 7 mEq/L → dialysis.
- If K < 7 mEq/L → IV sodium bicarbonate 100 ml IV infusion.
- Glucose 25% 100 cc + 20 unit regular insulin infusion over 3 hours.

→ Beta-2 stimulation by injection or inhalation.

* IV Calcium gluconate as calcium antagonist K effect on the heart

TTT of ↑↑↑K BIG C

- Bicarbonates
- Insulin
- Glucose
- Calcium



average PCO_2 (40)
 40 mmHg
 7.38
 0.08
 7.38

Metabolic Acidosis
 (Shock)
 pH $\downarrow$
 HCO_3 $\downarrow$

Metabolic Alkalosis
 (p. obstructive
 vent)
 pH $\uparrow$
 HCO_3 $\uparrow$

Resp. acidosis
 (K. failure)
 pH $\downarrow$
 HCO_3 $\uparrow$

Respiratory compensation
 islate
 H_2O
 H_2O
 Acute

Resp. alkalosis
 pH $\uparrow$
 HCO_3 $\downarrow$

Acid-Base Regulation

⇒ In brief:

- The products of metabolism are predominantly acids {CO₂ & organic acids}.
- Maintenance of stable pH is initially achieved by buffer systems (HCO₃⁻ [is the most important as it is **easy manipulated by lung & kidney**], Hb & protein).
- pKa is the pH of a buffer at which half the acid molecules are undissociated and half are associated.

$$pH = pKa + \log \frac{HCO_3^-}{H_2CO_3}$$

- Acidaemia is arterial blood pH of <7.35
- Alkalaemia is arterial blood pH of >7.4
- Acidosis is abnormal condition demonstrated by a decrease in arterial pH.
- Alkalosis is abnormal condition demonstrated by an increase in arterial pH.

► Body buffer systems:

- Consists of salt of weak acids (bicarbonate, hemoglobin, tissues and bones).
- The most important is bicarbonate carbonic acid ratio $\frac{HCO_3^-}{H_2CO_2}$

Blood pH and electrolytes can be assessed by arterial blood sample

Normal ranges of ABG:

▪ H ⁺	36-44	mmol/L
▪ HCO ₃ ⁻	22-26	mmol/L
▪ pO ₂	75-100	mmHg
▪ pCO ₂	35-42	mmHg

❖ Anion gap:

Cations	Mmol/l	Anions	Mmol/L
Na ⁺	140	Cl ⁻	105
K ⁺	5	HCO ₃ ⁻	25
Total	145	Total	130

- There is a gap between anion & cation due to uncalculated anions
- Anion gap value = 10 – 15.
- Some cases of acidosis due to organic acid production as DKA, has wider anion gap, because we do not measure this anion.
- Some cases of acidosis with normal anion gap as cases of diarrhea → there is loss of HCO₃⁻, but there is Cl⁻ retention by the kidney as Cl⁻ is measured.

★ How to deal with a problem solving, in examples of PH changes

	CO ₂	pH	HCO ₃ ⁻
Respiratory acidosis	↑	↓	↑
Respiratory alkalosis	↓	↑	↓
Metabolic acidosis	No change	↓	↓
Metabolic alkalosis	No change	↑	↑

➤ In respiratory acidosis & alkalosis every 10 mmHg changes in PCO₂ (N = 40), will cause change 0.08 in PH.

Examples of MCQ questions

- Twenty-four hours after colon resection, urine output in a 70-year-old man is 10 mL/h. Blood chemistry analysis reveals sodium, 138 mEq/L; potassium, 6 mEq/L; chloride, 100 mEq/L; bicarbonate, 14 mEq/L. His metabolic abnormality is characterized by which of the following?
 - Abdominal distension
 - Peaked T waves *decreased* $\uparrow$ K^+
 - Narrow QRS complex
 - Cardiac arrest in systole *if* $\uparrow$ Ca^{2+}
 - J wave or Osborne wave
- A 24-year-old woman has acute renal failure following postpartum hemorrhage. Laboratory studies showed serum glucose, 150 mg/dL; sodium, 135 mEq/L; potassium, 6.5 mEq/L; chloride, 105 mEq/L; and bicarbonate, 15 mEq/L. Therapy should include which of the following?
 - Decrease potassium chloride to 10 mEq/L
 - Intravenous 0.9% sodium chloride
 - 100 mL of 50% glucose water with 10 U insulin
 - Intravenous calcitonin
 - Intravenous magnesium sulfate

if $\text{K}^+ > 7$ $\rightarrow$ dialysis
- A 55-year-old man with Crohn's disease had undergone resection of small bowel and anastomosis. Ten days later, he is found to have bilious drainage of 1 L/d from the drains. He is started on total parenteral nutrition (TPN). Four days later, his arterial blood gases (ABGs) are pH, 7.25; PO_2 , 98 mm Hg; and PCO_2 , 40 mm Hg. His anion gap is 10. The most likely cause is which of the following?
 - Diabetic ketoacidosis
 - Renal failure
 - Hypovolemic shock
 - Small-bowel fistula $\rightarrow$ *bec*, $\rightarrow$ *acidosis* $\oplus$ *Normal anion gap* $\rightarrow$ *Bicarb*
 - Uncompensated metabolic alkalosis

*Diarrhea $\rightarrow$ *metabolic acidosis**
- A 55-year-old man sustains numerous injuries involving the abdomen and lower extremities. During the intra- and postoperative periods, he is resuscitated with 10 L of Ringer's lactate and 2 U of packed red blood cells (RBC). After initial improvement, he has severe dyspnea on the second postoperative day. The most useful initial diagnostic test is which of the following?
 - Electrocardiogram
 - Analysis of arterial blood gas *✓*
 - Insertion of a central venous line
 - Ventilation-perfusion scan *✓* *Reveals bc*
 - Computed tomography (CT) scan of abdomen

MOF
- A 24-year-old woman is scheduled for an elective cholecystectomy. The best method of identifying a potential bleeder is which of the following?
 - Platelet count
 - A complete history and physical examination *✓*
 - Bleeding time
 - Lee-White clotting time
 - Prothrombin time (PT)

- following:
- | 120 i.e. vs Nat | 11 p/b |
|-----------------|--------|
| hypertonic | |
| saline | |

- 20 mEq
- 200 mEq
- 400 mEq
- 720 mEq
- 120 mEq

- | | Na | K | Cl | HCO ₃ |
|----|-----|----|-----|------------------|
| a. | 10 | 26 | 10 | 30 |
| b. | 60 | 10 | 130 | 0 |
| c. | 140 | 5 | 104 | 30 |
| d. | 140 | 5 | 75 | 115 |
| e. | 60 | 30 | 40 | 40 |

- | | Na | K | Cl | HCO ₃ |
|----|-----|---|-----|------------------|
| a. | 130 | 4 | 109 | 28 |
| b. | 154 | 0 | 154 | 40 |
| c. | 77 | 0 | 77 | 0 |
| d. | 167 | 0 | 0 | 167 |
| e. | 513 | 0 | 513 | 0 |

(Q 10 - 11)

A 70-year-old man has undergone anterior resection for carcinoma of the rectum. He is extubated in the operating room (OR). In the recovery room, he is found to be restless with an HR of 136 bpm and a BP of 144/80 mm Hg. ABG analysis on room air reveals pH 7.24; PCO_2 60 mm Hg; PO_2 54 mm Hg; HCO_3^- 25 mEq/L; and SaO_2 90%.

Handwritten notes:

- Arrows point from "restless" to "HR" and "BP".
- A box contains "20 L/min" with an arrow pointing to "room air".
- A box contains "Acute" with an arrow pointing to "Normal".

- Respiratory alkalosis
- Respiratory acidosis

11. Appropriate management for this patient should be which of the following?

- a. To administer 40% oxygen by mask
 - b. Morphine, 2 mg IV
 - c. Ringer's lactate, 250 mL over 1 hour
 - d. Intubation and ventilatory support
 - e. Deep breathing and coughing
- Resp. Failure*

12. A 60-year-old woman with mild hypertension is admitted for elective hysterectomy. On preoperative evaluation, she is found to have osteoarthritis; over the previous 6 months, she had noted watery diarrhea that was becoming progressively worse. The serum potassium is 3 mEq/L. Which is the most likely cause of hypokalemia?

- a. Myoglobinemia
- b. Villous adenoma of colon
- c. High-output renal failure
- d. Massive blood transfusion
- e. Spironolactone (Aldactone)

(Q 13 & 14)

A 64-year-old man underwent major abdominal surgery to remove a ruptured aortic aneurysm. Four days after the operation, an attempt was made to wean him off the ventilator. ABG analysis reveals pH, 7.54; PCO₂, 30 mm Hg; PO₂, 110 mm Hg; HCO₃, 30 mEq/L; and SaO₂, 99%.

13. Blood gas analysis reveals which of the following?

- a. Respiratory acidosis
- b. Metabolic alkalosis
- c. Respiratory alkalosis
- d. Compensated respiratory acidosis
- e. Combined respiratory and metabolic alkalosis

14. What will be the most likely complication due to the metabolic changes experienced by the patient?

- a. Hypokalemia
- b. Shift of oxyhemoglobin dissociation to the right
- c. Hyperkalemia
- d. Hypercalcemia
- e. Hyperchloremia

(Questions 15 - 17)

A 27-year-old man is involved in a car crash while traveling in excess of 70 mi/h. He sustains an intraabdominal injury and a fracture of the femur. The BP is 60/40 mm Hg, and the hematocrit is 16%.

15. Which physiologic changes will ensue?

- a. Peripheral vasodilation
- b. Inhibition of sympathetic tone
- c. Temperature rise to 103.8°F
- d. Eosinophilia
- e. Lactic acidosis

16. There is likely to be a proportionately greater increase in blood flow to which of the following?

- a. Kidneys
- b. Liver
- c. Heart
- d. Skin
- e. Thyroid gland

17. Initial resuscitation is best done by administration of which of the following?

- a. D₅W
- b. D₅W and 0.45% normal saline
- c. Ringer's lactate solution
- d. 5% plasma protein solution
- e. 5% hydroxyethyl starch solution

18. A 30-year-old man is brought to the emergency department following a high-speed car accident. He was the driver, and the windshield of the car was broken. On examination, he is alert, awake, oriented, and in no respiratory distress. He is unable to move any of his four extremities; however, his extremities are warm and pink. His vital signs on admission are HR 54 bpm and BP 70/40 mm Hg. What is the diagnosis?

- a. Hemorrhagic shock
- b. Cardiogenic shock
- c. Neurogenic shock
- d. Septic shock
- e. Irreversible shock

(Questions 19 - 22)

A 48-year-old man with severe vomiting as a result of gastric outlet obstruction is admitted to the hospital. There is marked dehydration, with urine output 20 mL/h, and the hematocrit is 48%.

19. Clinical confirmation of pyloric obstruction is most readily established by which of the following?

- a. Observation of peristalsis from left to right
- b. Observation of peristalsis from right to left
- c. Percussion of the upper abdomen
- d. Succussion splash
- e. Auscultation of the upper left abdomen

20. What is the predominant metabolic abnormality?

- a. Aspiration pneumonia with respiratory alkalosis
- b. Hypochloremic alkalosis
- c. Salt-losing enteropathy
- d. Intrinsic renal disease
- e. Metabolic acidosis

21. Initial treatment for this patient should include which of the following?

- a. Administration of 10% dextrose (D₁₀W) in one-third saline solution IV
- b. Antiemetic
- c. Hemodialysis to correct azotemia
- d. Saline fluid replacement with appropriate potassium administration
- e. Ringer's lactate solution

22. In the absence of malignancy, further treatment after appropriate resuscitation should include which of the following?
- Jejunostomy feeding
 - Vagotomy and drainage
 - Steroids
 - No foods given orally (PO) for 6 weeks
 - Pyloromyotomy alone

(Questions 23 - 25)

During cholecystectomy in a 67-year-old woman, there is severe bleeding from accidental injury to the hepatic artery. The patient requires transfusion of 2000 mL of blood. After the operation, 24-hour urine output varies between 1250 and 2700 mL/d. She was adequately hydrated, but BUN levels continue to rise 10-12 mg daily over a 5-day period.

23. What is the main finding?
- Progressive bleeding
 - High-output renal failure
 - Postcholecystectomy syndrome
 - Glomerulonephritis
 - Obstructive jaundice

24. Metabolic changes likely to occur include which of the following?

- Hyperkalemia
- Hyponatremia
- Hypophosphatemia
- Metabolic alkalosis
- Hypomagnesemia

25. Management includes which of the following?

- Restriction of fluids to 750 mL/d
- 8 L of fluid daily to remove urea
- Replacement of fluid loss plus insensible loss
- 80 mEq potassium chloride (KCl) per 12 hour
- Ammonium chloride IV

26. In metabolic alkalosis, there is which of the following?

- Gain in fixed acid
- Loss of base
- Hyperkalemia
- Rise in base excess
- Hyperchloremia

Answers

1.	B	7	D	13	E	19	D	25	C
2.	C	8	C	14	A	20	B	26	
3.	D	9	A	15	E	21	D		
4.	B	10	B	16	C	22	B		
5.	B	11	D	17	C	23	B		
6.	B	12	B	18	C	24	A		

CHAPTER: 10

TUMORS IN SURGERY (ONCOLOGY)

- 1- General scheme for malignancy
- 2- Biology of cancer
- 3- Tumor markers
- 4- Radiation therapy
- 5- Cytotoxic chemotherapy

Tumors in Surgery (Oncology)

Definition

- Tumor is autonomous, excessive, purposeless and pathological proliferation of cells that continues indefinitely in absence of physiological stimuli.

General Scheme of Malignant Tumors

Predisposing Factors

1. **Idiopathic.**
2. **Genetic factors:** e.g.

Syndrome	Inheritance	Associated tumors
Familial polyposis coli & Gardner syndrome	Dominant	Colorectal cancer Osteoma of the jaw Desmoid tumor
MEN-I	Dominant	Parathyroid tumors Pancreatic tumors Pituitary tumors
MEN-IIa	Dominant	Medullary carcinoma of thyroid Pheochromocytoma Parathyroid tumor
MEN-IIb	Dominant	Medullary carcinoma of thyroid Pheochromocytoma Neurofibroma
Li-Fraumeni syndrome (P53)	Dominant	Leukemia Osteosarcoma Brain tumors
Familial breast cancer (BRCA-I & BRCA-II)	Dominant	Breast cancer Ovarian cancer Prostate cancer

3. Environmental factors:

- **Chemical:**
 - Tobacco: lung cancer, head and neck cancer and bladder cancer.
 - Alcohol: head and neck cancer, esophageal cancer and hepatoma.
 - Asbestos: mesothelioma of lung.
 - Aflatoxin: hepatoma.
- **Physical:**
 - UV-rays: melanoma and non-melanoma skin cancers.
 - Exposure to irradiation.
 - Mechanical irritation: e.g. gall bladder stones.
- **Infection:**
 - Viral: HPV (cervix and penile cancer), HIV (Kaposi sarcoma, lymphoma and anal cancer) and HBV or HCV (hepatoma).
 - Bacterial: H. pylori (cancer stomach).
 - Parasitic: Bilharziasis (cancer bladder).

4. **Obesity**: associated with tumors in the breast, endometrium, colon & esophagus.

5. Pre-malignant tumors.

Incidence

More common in elderly males

Pathology

- Site: according to type of tumor
- Macroscopic picture: according to site of tumor:-
 - **In solid organs** → star-shaped mass with infiltrating edges + areas of hemorrhage & necrosis.
 - **In hollow organs** → ulcerative, cauliflower mass or infiltrative lesion.
- Microscopic picture: according to lining epithelium of tumor + criteria of malignancy (Pleomorphism & mitotic figures, acidophilic cytoplasm and acidophilic nuclei).
- Stages of cancer development:
 1. Hyperplasia
 2. Metaplasia
 3. Dysplasia
 4. In situ carcinoma
 5. Invasion
 6. Metastasis

Spread

- **Direct (Local) spread**: to adjacent tissues.
- **Lymphatic spread**: to draining L.Ns.
- **Blood spread (2L2B)**: liver, lung, bone & brain.
- **Transcoelomic spread**: in GIT malignancies:
 - ⇒ When the tumor reaches the serosa → malignant cells spread toward the peritoneal cavity → peritoneal nodules + malignant ascites → then to pelvis → Krukenburg tumor + nodules in Douglas pouch.

Clinical Picture

Malignant tumors may cause:

- Rapidly progressive system effect.
- Predominant local effect

1. Local effect of tumors: varies according to site:

- a) Tumors on surface of the body → may bleed or discharge.
- b) Tumors in a hollow viscus or duct → obstruction, bleeding, discharge, perforation.
- c) Tumors in closed space →
 - Pressure symptoms (e.g. ↑ ICT in brain tumors)
 - Invasion of the organ → affect the function of the organ → organ failure.
 - Invasion of nerves → severe pain.

2. Systemic effect of tumors

A. Generally → cachexia, anemia, weight loss and fever of unknown origin (FUO).

B. Spread

- a. Direct: to nearby organs.
- b. Blood: mainly to :
 - 1) Brain → ↑ ICT, focal neurological manifestations.
 - 2) Lung → infiltration, cavitations, infection and pleural effusion.
 - 3) Bone → bone ache and pathological fractures.
 - 4) Liver → pain in Rt. hypochondrium, jaundice, enlarged hard tender liver.

- c. Lymphatic spread:
 - 1) Enlargement of regional lymph nodes.
 - 2) Later on enlargement of supraclavicular LNs (Troisier's sign).
- d. Transcoelomic spread: by seeding or by retrograde lymphatic spread.
- C. **Some tumors may cause non-metastatic (paramalignant) manifestations**
 - a. Clubbing & hypertrophic osteoarthopathy.
 - b. Neurological manifestation:
 - Peripheral neuropathy, myopathy.
 - Polymyositis, dermatomyositis and myasthenia gravis.
- D. **Secretion of abnormal proteins may cause characteristic syndrome**
 - a. Appropriate secretion where the tumor cells secretion is related to tumor site e.g. tumor of adrenal cortex → excess cortisone → Cushing disease.
 - b. Inappropriate secretion not related to site of tumor e.g. Bronchogenic carcinoma → ↑ ACTH → stimulate normal adrenal gland → ↑ cortisone level.

Staging

- **Stage I** → Tumor is localized to the organ.
- **Stage II** → Tumor with enlarged mobile draining LNs.
- **Stage III** → Tumor with enlarged fixed draining LNs (± local invasion).
- **Stage IV** → Tumor with distant metastasis.

TNM classification of malignant tumor:

It is a cancer staging system that describes the extent of cancer in a patient's body. **T** describes the size of the tumor and whether it has invaded nearby tissue, **N** describes regional lymph nodes that are involved, and **M** describes distant metastasis.

Investigation of malignant tumors

Investigations

For diagnosis

For staging

For preoperative preparation

For Follow up

For screening

- **For diagnosis**: according to type of tumor.
- **For staging**:
 - 1. **Sentinel Lymph Node Study**:
 - By injection of methylene blue or radioactive isotope → follow-up → till we find the sentinel lymph node.
 - Then it is excised and frozen section is done for it to know whether affected by the cancer or not.
 - 2. **Lung** → CXR.
 - 3. **Liver** → abdominal ultra sound and liver function tests.
 - 4. **Bone** → bone scan (Tc 99).
 - 5. **Brain** → CT scan and MRI.
- **For pre operative preparation**:
 - CBC, FBS, LFTs, KFTs.
- **For follow up**:
 - 1. **Tumor markers**: CA15-3 and CEA.
 - 2. **Biochemical investigations (hormonal receptors)**: estrogen and progesterone.
- **For screening**: as in
 - 1. Mammography in cancer breast
 - 2. Calcitonin level in medullary carcinoma of thyroid.

Treatment**1. Surgery.**

- ▣ **Primary tumor:** radical surgery aims at excision of the primary tumor with as wide safety margin.
- ▣ **Lymph nodes:** the treatment of lymph nodes varies from one tumor to another.
- ▣ **Precautions:** during surgery care is taken to avoid spillage of malignant cells.
- ▣ **Advantages:** surgical excision is both quick and effective for the largest number of cures to confirm that a tumor has been fully excised.
- ▣ **Disadvantages:** may produce functional and cosmetic disabilities, it can't be applied if the tumor is fixed to a vital structure or if has produced distant spread.

2. Radiotherapy

- ▣ May replace surgery or may be given in addition.
- ▣ Common indications. Cancer of the larynx so as to preserve the voice, early Hodgkin's disease, early prostate cancer, and as a part of conservative therapy for early breast cancer(after surgery).
- ▣ **Methods:** powerful x ray, gamma rays, electrons, or heavy particles.
- ▣ **Advantages.**
 - a. Can preserve the anatomical structures that surround a cancer.
 - b. Radiation can destroy microscopic extensions of cancerous tissue around a tumor.
 - c. Is a safer option for older, frailer patients who might have difficulty recovering from surgery.
 - d. Patients treated with radiation usually don't require hospitalization.
- ▣ **Disadvantages:**
 - i. In general is much less sensitive.
 - ii. Burns of the skin or enteritis.
 - iii. Compared to surgery ,radiotherapy is slower as it usually takes five to eight weeks ,not suitable for metastatic cases .

3. Chemotherapy:

- ▣ **Common Indications:** Is the main ttt of leukemias.
- ▣ Better results are obtained from combination chemotherapy.
- ▣ **Advantages:** travel through circulation, can reach malignant cells anywhere.
- ▣ **Disadvantages:** often kill many healthy cells and thus bring on serious side effects, tendency to damage to the rapidly growing cells of the bone marrow.

4. Hormone therapy: has relatively mild side effects because its actions are limited largely to tissues with receptors for specific hormones.**5. Immunotherapy:**

- ▣ **Non specific:** BCG induce remission in cases of transitional cell carcinoma of the urinary bladder.
- ▣ **Specific:** monoclonal antibodies from a single clone of lymphocytes.
- ▣ **Bone marrow transplantation:** not a therapy in itself but is sometimes used to strengthen the depleted bone marrow (blood-making system).

6. Biological therapy:

- ▣ Tremendous advances in oncology and molecular biology allowed the emergence of newer anti-cancer drugs which antagonize certain biological agents expressed by certain tumors as the next examples:

Drug	The biological agent antagonized
Herceptin: for breast cancer	Her-2 neu receptors.
Gleevec (imatinib): for gastro intestinal stromal tumors, GIST	Tyrosine kinase

	Early cancer (Potentially curable)	Advanced cancer (Incurable)
Definition	stage I or II	stage III or IV
Aim	Cure	Palliation
Disease status	Mainly local disease ± micro-metastasis	Mainly systemic disease
Primary treatment	Surgery ± radio-therapy (Local treatment)	Chemotherapy & endocrinal therapy (Systemic treatment)
Adjuvant treatment (Palliative)	Endocrinal & chemotherapy	Simple mastectomy & radio-therapy have limited role in local control

Tumor Markers

Definition

- Some malignant tumors produce substances (enzyme, hormone, protein or antigen) that can be detected in the blood and may be used as marker of both the presence of the tumor and screening for early recurrence following treatment.
- Tumors markers are also useful in assessing the response of Tumors to treatment.

☞ **An ideal tumor marker should have the following characteristics:**

- Always produced by the tumor.
- Gives a sensitive indication of the tumor mass.
- Produced by recurrent and metastatic disease.
- Specific for the disease and easy to measure.
- The tumor should be treatable.

Clinical use of tumor markers

Marker	Cancer	N.B.
CA 15-3	❖ Breast cancer	
Calcitonin	❖ Medullary carcinoma of thyroid	• If high total thyroidectomy is indicated even if thyroid clinically & by investigations is normal
Thyroglobulin	❖ Follicular & papillary thyroid carcinoma	
Human chorionic gonadotrophin (β-HCG)	❖ In both seminoma & teratoma	• AFP: - Normal level (0-10 mg/dl).

α -Feto-protein (AFP)	❖ Hepatoma (HCC) ❖ Only in teratoma, never in seminoma	- > 500 mg/dl → is highly suggestive of malignancy. - It is non-specific & may rise in: 1. Pregnancy. 2. Liver cirrhosis. 3. Chronic hepatitis.
LDH	❖ In seminoma (60% of cases) ❖ Also, in neuroblastoma	• These 3 markers are used for: 1. Diagnosis 2. Prognosis 3. Follow up after surgery
Carboxy-prothrombin	❖ HCC ❖ Fibrolamellar type of hepatoma.	• More specific
Carcino - Embryonic Antigen (CEA)	❖ Cancer stomach ❖ Cancer pancreas ❖ Cancer Breast ❖ Cancer colon	• > 65% indicates extensive spread
CA19-9	❖ Cancer stomach ❖ Pancreas	
CA 72-4	❖ More recent & specific for cancer stomach	
POFA (pancreatic oncofetal Ag)	❖ Cancer pancreas	
PCA (Pancreatic Cancer Associated Ag)		
5-HIAA (5-OH Indol Acetic Acid in urine)	❖ Cancer appendix	
VMA (Valinyl Mandelic Acid in urine)	❖ Pheochromocytoma	
Nuclear matrix protein 22	❖ Cancer urinary bladder	• For screening

Acid phosphatase	❖ Cancer prostate	
PSA (Prostatic specific Ag)	❖ Cancer prostate	• More specific than acid phosphatase. • It is a serum protease helping in liquefaction of semen. • Normally 0-4 ng/ml. • If > 30 ng/ml → mostly metastatic cancer prostate. • If < 15 ng/ml → may be BPH or cancer prostate.
CA 125	❖ Cancer ovary	
Enolase +ve	❖ Pheochromocytoma. ❖ Neurofibromatosis. ❖ Wilm's tumor.	
KRAS	❖ Recently in most of GIT tumors & cholangiocarcinoma	
Alkaline phosphatase	❖ Osteosarcoma	
PAP (Placental Alkaline Phosphatase)	❖ Cancer ovary. ❖ Seminoma.	

Radiation Therapy

Definition

- It is the treatment of malignant tumors with ionizing radiation, which is composed of high energy rays that cause ejection of atomic electron when absorbed.

Indications

- Curative:** used alone in some tumors e.g. Hodgkin's disease.
- Neoadjuvant:** for down-staging of the tumor before surgery.
- Adjuvant:** for radical removal of tumor residuals after surgery.
- Palliative:** if the case is inoperable and irresectable.

Mechanism of Radiation Therapy

- The effects are due to production of highly reactive O_2 free radicals.
- Effect is enhanced in the presence of molecular oxygen.
- The radicals interact with molecules in the irradiated cells $\rightarrow$ change molecular structures.
- Such changes may damage cellular DNA or the protein mechanism needed for cellular reproduction and metabolism.

Radio sensitivity

- Is the susceptibility of a certain cell to be damaged by ionizing radiation rapidly dividing cells tend to be much more radiosensitive than slowly dividing ones.
- Types:**
 - Curable:** e.g. rodent ulcer.
 - Resistance:** e.g. adenocarcinoma.
 - Sensitive:** e.g. Wilm's tumors.

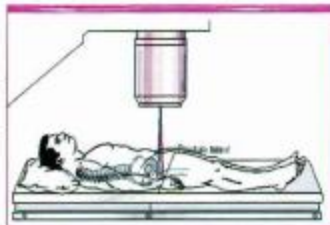
Dose

- Doses are measured in gray (Gy) units, which are equal to the absorption of 1 joule /kg of tissue.
- One gray is equal to 100 rads.
- The probability of controlling a tumor with irradiation is inversely related to the size of the tumor.

Modes of delivery

1. External beam radiation teletherapy (EBRT):

- It is the most common mode of delivery.
- It uses a device located some distance from the patient to focus radiation on the target tissues.
- The goal is to deliver maximal radiation to the tumor while excluding normal surrounding tissue.



2. Brachytherapy:

- e.g. intestinal irrid. needle
- The principle is to place a radiation source near the site of the primary tumor either surgically or through the normal body openings e.g. in prostate cancer.
- Other methods involve the use of catheters that can be filled with radioisotopes.

3. Intra-operative radiation therapy:

- Used in the operating room after tumor resection.

4. Preoperative:

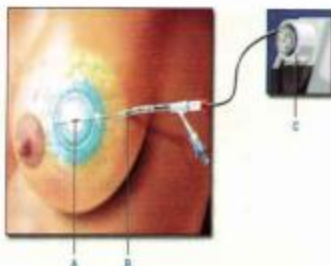
- e.g. in cancer rectum to change it from irresectable cancer into resectable cancer (down staging).

5. Post-operative**6. Systemic targeting:**

- Depends on unique features of the tumor cells, as in the specific uptake of radioactive I^{131} by certain types of thyroid cancers.
- **Advantage** → allows treatment of tumors which may have metastasized or spread beyond anatomic boundaries.

7. Radio-immunotherapy:

- It is an experimental mode of systemic delivery.
- Radioisotopes are bound to antibodies targeting tumor-specific antigens.
- This technique has shown great promise in the treatment of many cancers, including hepatocellular carcinoma and lymphoma.

**Disadvantages**

1. **Resistance** of some tumors to radiotherapy e.g. adenocarcinoma.
2. **Slower onset** of action when compared to surgery.
3. **Associated complications:** end arteritis obliterans, skin burn, ulcers, bone marrow depression, enteritis, intestinal obstruction or fistula formation, sterility, interstitial pulmonary fibrosis and radiation sickness.

Cytotoxic Chemotherapy

Definition

- It is treatment of malignant tumors using pharmacologic agents systemically.
- The ideal tumor drug would kill cancer cells without affecting normal tissues.

Basic principles of combined therapy

1. Use of effective agent.
2. Use agents with different mode of action (synergism).
3. Use agents with non-overlapping toxicity.
4. Consider spatial co-operation.

Indications

- **Curative:** used alone in some tumors e.g. leukemia.
- **Neoadjuvant:** for down-staging or shrinkage of the tumor before surgery.
- **Adjuvant:** for elimination of any microscopic foci of systemic malignancy.
- **Palliative:** if the case is inoperable and irresectable.

Classes of Chemotherapeutic Agents

- Chemotherapeutic drugs are generally classified as:
 - Cell cycle specific (CCS) drugs, which are toxic to actively proliferating cells;
 - Cell cycle-nonspecific (CCNS) drugs, which are capable of killing cells that are not dividing during drug exposure.
- These two classifications are not absolute, and many drugs may overlap between the two categories.
- Combination helps against tumor resistance and increase the tumor killing while avoiding the compounding of toxic effects.

	Mechanism	Uses	Examples
Alkylating agents	Breaking of DNA, miscoding or cross linking	- Hematological tumors - Some solid tumors e.g. breast – lung	Cyclophosphamide Cisplatin
Anti-metabolite	- Incorporate in nucleic acid - Shut down of cellular synthetic mechanism	- Hematological tumors - Some solid tumors e.g. breast – GIT	Methotrexate (MXT) 5-Fluorouracil
Antibiotics	- Blocks DNA synthesis - Breaking DNA strands - Anti-tumor activity	Solid tumors	Doxorubicin Dactinomycin
Plant alkaloid	- Vincristine → arrest mitosis in metaphase - Others (e.g. Etoposide) → topoisomerase enzyme inhibitors → inhibit cleavage and re-ligation of DNA	- Hematological tumors - Some solid tumors (breast – kidney – testis – head & neck cancers)	Vincristine Etoposide (derived from natural plants)

Side-effects of chemotherapy

- Metallic taste for 2-3 days after treatment.
- Nausea, vomiting, diarrhea.
- Rashes and ulceration of the skin.
- Hair loss (not all drugs cause this).
- Drug resistance** causes chemotherapy failures → combination therapy.
 - **Mechanisms**
 - Pumping drugs out of tumor cells.
 - Alteration of target enzymes.
 - Increased production of a target enzyme to overcome the drug.

Surgical Nutrition

Physiological consideration

- Normal person needs 30 Kcal/kg body weight (average 2100 Kcal/day).
- The energy given by each type of food:
 - 1 gm carbohydrate $\rightarrow$ 4 Kcal
 - 1 gm protein $\rightarrow$ 4 Kcal
 - 1 gm fat $\rightarrow$ 9 Kcal
- Calorie (CHO + fat) : nitrogen = 200 Kcal : 1 gm
- So, the ratio of the different items in a well-balanced diet:
 - carbohydrate $\rightarrow$ 50%
 - fat $\rightarrow$ 35%
 - protein $\rightarrow$ 15%
- Maintaining a healthy nutritional status requires the following daily balanced supplementation:

- **Etiology:** starvation, $\downarrow$ catabolism
- **Effects:** $\downarrow$ (healing, immunity, recovery, tolerance to radio & chemo)
- **Diagnosis:** anthropometric, lab.

	Per Kg	For 70 Kg person
- Water (ml)	35	2450
- Carbohydrate (gm)	2	140
- Fat (gm)	3	210
- Protein (gm)	0.7	50
- Nitrogen (gm)	0.7	7
- Na ⁺ (mmolL)	1	70
- K ⁺ (mmolL)	1	70

- Proteins are better assessed by its nitrogen contents.

- So, if the intake of nitrogen exceeds its amount in urine = (positive nitrogen balance) = anabolic state.

- If the reverse = (negative nitrogen balance) = catabolic state.

Malnutrition in surgical patient

Incidence

- About 30% of patients with GIT diseases.
- It reaches 60% in those with prolonged hospital stay.

Etiology

A. Starvation ($\downarrow$ input):

1. Social causes. as poverty & neglected elderly
2. Loss of appetite by chronic diseases.
3. Dysphagia.
4. Repeated vomiting.
5. Malabsorption syndrome $\rightarrow$ in {
 - Chronic pancreatitis
 - Short gut syndrome
 - Crohn's & ulcerative colitis
6. Post-operative restriction of oral intake (e. g. paralytic ileus).

B. Hypercatabolism ($\uparrow$ output):

1. Major trauma and burns.
2. Major surgical operations.
3. Severe acute pancreatitis.
4. Major sepsis e. g. septicemia.

Effects on the outcome of surgery

- > **Operative mortality & morbidity** ↑ when 20% of the body weight is lost.
1. Impairment of wound healing. e.g: burst abdomen
 2. Immunosuppression with ↑ susceptibility to infection.
 3. Delay physical recovery & ↑ hospital stay.
 4. Decreased tolerance to radiotherapy and chemotherapy.

Diagnosis

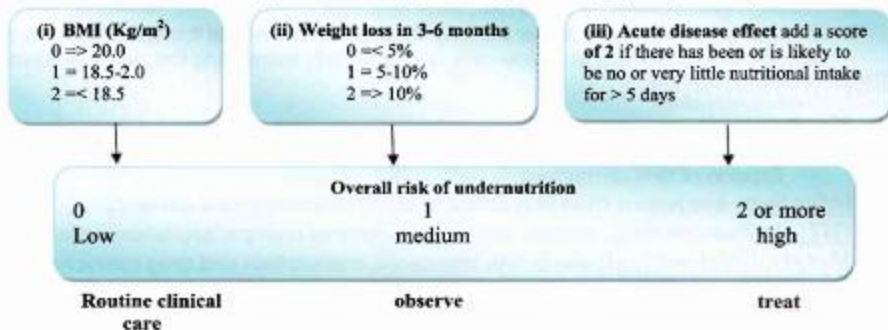
A. Anthropometric measures:

1. Recent un-intentional **weight loss** > 10%.
2. **Body weight** is < 80% of the ideal for height.
3. **Muscle mass:** Mid-arm muscle circumference < 80% of the average.
4. Triceps **skin fold** thickness.

B. Laboratory:

1. Serum albumin < 3.5 gm/L.
2. Determine nitrogen balance (whether +ve / -ve).
Input – output → +ve nitrogen balance (anabolism).
→ -ve nitrogen balance (catabolism).
3. TLC < $1.200 \times 10^9/L$.
4. Impaired hypersensitivity reaction.

The MUST tool: the British association of parenteral and enteral nutrition introduced mal-nutrition universal screening tool (MUST)



Types of Surgical nutrition

A. Enteral nutrition.

B. Parenteral nutrition.

A. Enteral Nutrition

Definition

- Delivery of nutrients into the GIT.

Indications

- Patient in whom oral intake is inadequate whenever there is a functional accessible
- GIT (as comatosed patient, severe dysphagia, neck surgery, low output GIT enterocutaneous fistula and burns).

Routes of administration

1- **Sip feeding:** whole food by mouth (fluid formula).

2- **Tube feeding techniques:**

- Nasogastric tube (NGT):** Ryle's tube
- Gastrostomy:** if nasogastric feeding is not possible e. g. upper GIT obstruction.
- Jejunostomy:** if the stomach is diseased.

Administration formulae

A- Through gastrostomy → liquid diet, juice and milk.

B- Through jejunostomy → either partially digested or elemental formulae, starting with isotonic sterile formula at a slow rate, and gradually increasing the rate and tonicity.

Complications

1- **Mechanical:**

- Malposition, displacement, blockage, breakage or leakage of the tube.
- Erosion of skin or mucosa.

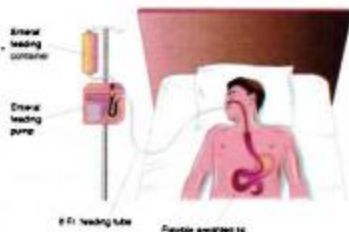
2- **Infective:** exogenous (handling contamination) or endogenous (patient).

3- **GIT:** diarrhea, bloating, nausea, vomiting, abdominal cramps, aspiration, constipation.

4- **Metabolic/chemical:** electrolyte imbalance, malnutrition and drug interactions.

> Tube Care:

- Flushed with water at least twice per day.
- If obstructed with solidified diet → instillation of chemotrypsin inside the tube may salvage a partially obstructed tube.



B. Total Parenteral Nutrition (TPN)

Definition

- Provision of all nutritional requirements through intravenous route without use of GIT.

Indications

- The principle indication for TPN is found in seriously ill patients suffering from protein calorie malnutrition where the use of GIT for feeding is not possible as follow:
 - 1- GIT is **blocked** e.g. stricture, neoplasm or extrinsic mass.
 - 2- GIT is **short** e.g. short gut syndrome.
 - 3- GIT is **fistulated** e.g. enterocutaneous fistula.
 - 4- GIT is **inflamed** e.g. inflammatory bowel disease.
 - 5- GIT can **not cope** e.g. severe trauma or hyper-catabolic state.
 - 6- Radiation enteritis.
 - 7- Moderate to severe pancreatitis.
 - 8- Paralytic ileus.

Route of Administration

- Central:** catheter inserted via subclavian or internal or external jugular vein.
- Peripheral:** (appropriate for short term feeding up to 2 weeks)
 - iv. Peripherally inserted central venous catheter (PICC) line.
 - v. Conventional short cannula in the wrist vein (isotonic fluid).

Calculation of requirements

- Normal person needs 30 Kcal/Kg body weight, 2400 Kcal/day.
- Surgical patient need 40 Kcal/Kg body weight.
- The energy given by each type of food:
 - 1 gm carbohydrate $\rightarrow$ 4 Kcal
 - 1 gm protein $\rightarrow$ 4 Kcal
 - 1 gm fat $\rightarrow$ 9 Kcal
- Calorie (CHO + fat) : nitrogen = 200 Kcal : 1 gm
- So, the ratio of the different items in a well-balanced diet:
 - carbohydrate $\rightarrow$ 50%
 - fat $\rightarrow$ 35%
 - protein $\rightarrow$ 15%
- The requirement is given in 2-4 liter of fluids.
- Types of solutions used
 1. CHO $\rightarrow$ Glucose 50% + insulin.
 2. Fat $\rightarrow$ Intralipid 10% & 20%.
 3. Protein $\rightarrow$ vamine or totamine.
- Other elements which has to be added:
 1. K^+ , Mg^{+2} and Cl^-
 2. Trace elements.
 3. Water-soluble vitamins and fat soluble vitamins.
- The fluids are mixed together in a mixer bag & given over 24 hours in CVP.
- Lipids can be given through a peripheral vein because it is isotonic

Important notes about TPN▪ **Intralipid:**

- Fat emulsion produced from soya oil.
- It is isotonic.
- Given in peripheral vein.
- Not to mix it in the same mixture bag.
- Given twice weekly.

▪ **CHO:**

- Raise the dose every 3 – 4 days, regards to ↑ insulin output gradually.

Complications

- Nutritional and metabolic complications resulting in overfeeding, underfeeding or specific nutrient imbalance as hyponatremia, hypokalemia or hyperosmolar dehydration. Hyperglycemia is a common incident.
- **Catheter complications:**
 1. Misplacement of the catheter.
 2. Insertion of a catheter tip in the pleural cavity leading to hemothorax or pneumothorax. Chest x ray should always be done after insertion of a catheter.
 3. Injury to arteries or to nerves of brachial plexus ,rare in experienced hands.
 4. If the infusion set is accidentally detached from the venous catheter air can be sucked into the superior vena cava which have a negative pressure producing serious air embolism.
 5. Venous thrombosis.
 6. A common serious complication is the central venous catheter infection & septic thrombophlebitis which lead to septicemia and if neglected , death, whenever the patient develops unexplained fever that persists for more than 24 hours the venous catheter should be removed and sent for bacteriological and fungal cultures.
- Failure of blood barrier, the absence of enteral feeding leading to atrophy of intestinal mucosa. This allows for translocation of bacteria from the lumen to the blood stream leading to septicemia in the critically ill patients.

Monitoring of the patient

- **Daily:** → urine output, Blood (Urea, Electrolyte, Glucose).
- **Twice weekly:** → Ca, Ph, Albumin, CBC
- **Weekly:** → LFT, Plasma lipids, Magnesium.
- **Monthly:** → foliate, Iron, Zinc, PT

Refeeding Syndrome:

- Severe fluid and electrolyte shift in malnourished patient undergoing refeeding, more common with TPN, but may occur with enteral nutrition.
- Manifested by ↓ Ca, Mg and P → arrhythmia, liver dysfunction, seizures, confusion, ↓ respiratory function, coma up to death.
- Risk patient: alcohol dependant patients, severely malnourished patients.
- Treatment: matching the intake with requirements, avoid overfeeding, regular vitamin administration, Mg and P supplementation.

C. Home Parenteral Nutrition

- Intravenous nutrition at home is usually reserved for patients who had massive small intestinal resection (short bowel S).
- 1. Permanent silastic central venous catheter, tunneled & cuffed is placed for long-term use.
- 2. Parenteral alimentation, is usually given at night over 12 hours with the aid of mechanical pump.

➡ Metabolic response to starvation:

1. Low plasma insulin.
2. High plasma glucagon.
3. Hepatic glycogenolysis.
4. Protein catabolism.
5. Hepatic gluconeogenesis.
6. Lipolysis: mobilization of fat stores.
7. Adaptive ketogenesis.
8. Reduction in resting energy expenditure.

➡ Metabolic response to trauma & sepsis:

1. ↑ Counter regulatory hormones:
(adrenaline, nor-adrenaline, cortisol, glucagon, growth hormone).
2. ↑ Energy requirements.
3. ↑ Nitrogen requirements.
4. ↑ Gluconeogenesis & protein catabolism
5. Insulin resistance & glucose intolerance.
6. Fluid retention & hypoalbuminemia.

Obesity in Surgery

Definition

- Basal metabolic index (BMI) of more than 30.

Data obtained during recent years shows that the distribution of fat is of greater importance than is the magnitude of obesity. People whose deposits of fat are in the trunk or abdomen (android obesity) have greater morbidity and mortality rates than those with a more typically female distribution on the hips and buttocks

Surgery

Problems of surgery in obese

- **Increased risk of:**
 - Difficult intubation & Aspiration.
 - Myocardial infarction, cerebrovascular accident and DVT.
 - poor wound healing and infection.
 - Bed sores.
 - Mechanical problems (lifting, transferring, operating table & weight limits).

Indications of surgical treatment for obesity

- 1) severely obese (BMI of 40 or more)
- 2) have a BMI of 35 to 39.9 with serious medical conditions (such as high blood cholesterol and triglycerides, hypertension, sleep apnea)
- 3) Tried other methods of weight loss (changes in eating, behavior, increased physical activity and/or drug therapy) and are still severely obese.
- 4) unable to physically perform routine daily activities (work-related and family functions)
- 5) Understand the procedure, risks of surgery and effects after surgery.

Operative procedures

Intestinal bypass and, to a certain extent, gastric bypass Operations limiting the amount of solid food that can be ingested or passed through the upper intestinal tract.

Types of operations

1. Small bowel resection:

Idea

- weight loss in patients with extensive resections
- The debility and hepatic dysfunction made this procedure obsolete.

2. Jejunioileal bypass

Idea

- A short-bowel syndrome can be created by joining the end of about 35 cm of proximal jejunum to the side of ileum about 10 cm proximal to the ileocolic junction.



End-side jejunioileostomy.

Complications:

- Hepatic dysfunction
- Hypokalemia .
- Hypocalcemia.
- Hypomagnesemia.
- Renal lithiasis from fat malabsorption.
- Oxaluria and dehydration.
- Cholelithiasis.
- A peculiar form of enteroarthritis.
- Prevalent diarrhea.

They are considered to be too high a price to pay.

3. Biliopancreatic bypass (not widely accepted).

4. Gastric bypass:

Idea

- A small gastric pouch is created by stapling across the stomach,
- The gastric pouch is drained by a Roux-en-Y gastrojejunostomy.

Complications:

- Deficiencies of iron, calcium, thiamine, vitamin B₁₂, and of folic acid
- Marginal (stomal) ulcers are rare complications.

5. Vertical banded gastroplasty:

- Aims to create both a small gastric pouch and an outlet that does not dilate.
- These goals are accomplished by placing four rows of staples parallel to the lesser curvature of the stomach, creating a gastric tube with a capacity of 15 ml or less.

Complications

- Perforations of the esophagus and stomach
- Mortality rates are generally below 1%.



Vertical gastroplasty

Results of antiobesity surgery

- Around 30 % of preoperative body weight is lost during the first 1 to 2 years.
- The walls of the stomach are smooth muscle, they expand with time.
- Compensatory hypertrophy and hyperplasia of the intestine increase absorptive capacity.
- Obese patients are at increased risk of postoperative complications such as wound infections, hernias, pneumonia, and thromboembolism.

Surgical Hypertension

➡ See in aid to post-graduate book.

Pheochromocytoma

➡ See in endocrine part

Cushing's Syndrome

➡ See in endocrine part

Renovascular Hypertension

➡ See in aid to post-graduate book.

Transplantation

Transplantation Terminology

Autograft:	Donor & recipient is the same person (no rejection).
Isograft:	Donor & recipient are identical twins (no rejection).
Allograft:	Donor & recipient are of same species but genetically different (human to human).
Xenograft :	Donor & recipient are of different species (animal to human).
Orthotopic graft:	A graft placed in its normal anatomical site
Heterotopic graft:	A graft placed in a site different from its normal anatomical site.
Alloantigen:	Transplant antigen
Alloantibody:	Transplant antibody

Immunity in transplantation

⇒ Major Histocompatibility Antigens:

- Glycoprotein on the surface of all somatic cells acts as "self markers" which are responsible for triggering the immune reaction leading to allograft rejection.
- These molecules were originally detected on leucocytes and therefore, named (Human Leukocyte Antigen, HLA).
- HLA are genetically controlled by loci on the short arm of chromosome 6, this area is termed the **major histocompatibility complex**.

⇒ ABO Antigens:

- These antigens are not only present on RBCs but also on most of other cell types.
- There is **no need** to consider **Rh** compatibility in transplantation.

Immune Compatibility Testing:

- ABO blood grouping and cross matching.
- HLA cross matching (**important** in kidney, bone marrow and pancreas transplantation while **not important** in heart and liver transplantation).

⇒ Mechanism of graft rejection:

- Cellular mechanism:** graft alloantigens → engulfed and processed by macrophages → stimulate T-helper lymphocytes → release interleukin-2 (IL-2) → stimulate cytotoxic T-lymphocytes → attack the transplanted tissues.
- Humoral mechanism:** cytotoxic antibodies (present before transplantation e. g. anti-A & anti-B) → complement fixation → attack vascular endothelium in transplanted organ → thrombosis (**hyper-acute graft rejection**).

⇒ Types of graft rejection:

Type	Onset	Mediator	Treatment	Result
Hyperacute	Immediate	Humoral ABO	Graft removal	Graft loss
Acute	First 6 months	Cellular	High dose steroid OKT3	Good
Chronic (commonest)	After 6 months	Cellular and/or humoral	None (irreversible) Or re-transplantation	Bad

Immune-suppressive therapy

- Immunosuppressive therapy is given to all transplant recipients in order to inhibit the rejection process.
- **Classification according to mode of action:**
 1. **Drugs which deplete circulating lymphocytes:** Corticosteroids, anti-lymphocytic globulin (ALG), Anti-thymocyte globulin (ATG) and monoclonal antibodies (OKT3).
 2. **Drugs which inhibit lymphocyte activation and/or proliferation:** Cyclosporine, tacrolimus (FK-506) and azathioprine.

The three major drugs are corticosteroids (prednisolone), azathioprine (imuran), and cyclosporine.

- **Complications:**
 - A. *Complications common for all drugs:*
 1. **Infection:** Bacterial, viral (the commonest is CMV), protozoal (the commonest is pneumocystis carinii) and fungal (candida).
 2. **Malignancy:** the commonest is squamous cell carcinoma of skin.
 - B. *Specific complications for each drug:* e. g.
 1. **Steroids** → DM, hypertension, weight gain, osteoporosis and peptic ulceration.
 2. **Azathioprine** → hepatotoxicity and leucopenia.
 3. **Cyclosporine & Tacrolimus** → nephrotoxicity.

Preparation for transplantation

Types of Organ donors

- **Cadaveric donors (brainstem-dead donors):**

- Brainstem death is caused by intracranial hemorrhage, trauma, viral/bacterial infection, or a primary intracerebral tumor.
- The diagnosis is confirmed by CT scan and a series of clinical tests performed twice by two senior doctors establish brainstem.
- The cadaveric donor should be monitored and supported to maintain the perfusion of tissues to be transplanted (ventilation, plasma and vasopressors).

- **Criteria of brainstem death:** in addition to the flat EEG the following should be checked:

- The patient must be in apneic coma: The patient is unresponsive & dependent on mechanical ventilation.
- No corneal reflex
- No pupillary response to light, direct or consensual
- Absent gag reflex
- Absent cough reflex via bronchial stimulation by a catheter in the endotracheal tube.
- Absent vestibulo-ocular reflex no eye movement upon injection of 50ml of ice cold water into each external auditory meatus.
- All the previous tests should be repeated after 24 hours and should be done by a separate physician not involved in the transplant procedure

- **Exclusions**

- **Drug effects:** as sedatives m hypnotics or muscle relaxants
- **Hypothermia:** core temp must be above 35 °C . Low body temperature can induce deep coma.
- **Metabolic abnormalities** e.g. hypo or hyperglycemia , hypo or hypernatremia , uremia or hepatic encephalopathy.
- **Intoxication** by alcohol or other drugs

- **Living donors:**

- As a healthy first-degree relatives (parent, child, or sibling).
- The donor should be examined, investigated, tissue typed and cross-matched with the recipient.

Organ recipient

- **Investigation of patients for suitability for organ transplantation:**

- CBC and coagulation profile and blood group.
- KFTs and LFTs.
- Glucose and lipid profiles.
- Chest X-ray and ECG.
- Viral markers for hepatitis B & C, HIV, CMV, HSV and EBV.
- **Tissue typing and antibody screening for HLA.**

- **Contraindications of organ transplantation:**

- Multi-organ failure.
- Ongoing sepsis.
- Current malignancy.
- Active peptic ulceration.

Specific organ transplantation

		<u>Kidney Transplantation</u>	<u>Liver Transplantation</u>	<u>Pancreas Transplantation</u>	<u>Bone Marrow Transplantation</u>
Indications		- <u>Commonest</u> is chronic glomerulonephritis - Chronic pyelonephritis - Lupus nephritis - Hypertension and DM - Polycystic kidneys - Advanced obstructive uropathy	- Cirrhosis (alcoholic, post-viral, biliary or Wilson's) - Biliary atresia (child) - Chronic viral hepatitis - Sclerosing cholangitis - Budd Chiari syndrome - Advanced malignancy	- Type I IDDM + renal failure	- Aplastic anemia - Some types of leukemia
Technique	Graft	Placed extra-peritoneal in the iliac fossa especially the Right Fossa	Placed in the same position (orthotopic transplantation)	Whole or segmental N.B. islets transplantation is still under trials	Bone marrow is aspirated from donor → filtration & heparinization Then, IV infusion to the recipient
	Anastomosis	• <u>Artery</u> → renal with external iliac (end-to-side) • <u>Vein</u> → renal with external iliac (end-to-side) • <u>Ureter</u> → to urinary bladder under mucosal tunnel	• Vessels and ducts are anastomosed to their corresponding	• <u>Veins</u> → to either portal or systemic circulation • <u>Ducts</u> → to the gall bladder or GIT (by Roux-en-Y)	
Complications		1. Anastomotic leakage or stricture 2. Rejection 3. Recurrence of original disease 4. Complications of immune-suppression (mention)			
Prognosis		1 YSR → 95% if cadaveric → 98% if living donor 5 YSR → 60% if cadaveric → 90% if living donor	1 YSR → 85% <u>5 YSR</u> → 50%	1 YSR → 45%	

Heart transplantation

- **Indications:** 1) Advanced CAD not suitable for coronary bypass. 2) Idiopathic cardiomyopathy
 (Combined Ht. & lung transplantation may be done if both organs are damaged)
- **Complications:** 1) Of immunosuppressive. 2) Graft rejection 3) Graft atherosclerosis

Imaging in Surgery

Ultrasound (SONAR)

= Sound on navigation and Ranging

Definition

- Sound waves at frequency between 2.5 - 7.5 MHz, which is more than what the human ear can hear.

Uses

- **Diagnostic:**
 - Easy, non-invasive and can be used Intra-operative (e.g. laparoscopic U/S).
 - Guided biopsy.
- **Therapeutic:**
 - Guided drainage.
 - Renal stones.

Disadvantages

- **Poor in:**
 - (Gas) e.g. colon → solved by laparoscopic US and endoscopic US
 - Fat people

Application

- 1- **Gall bladder:** calculi, shape, size, acute and chronic cholecystitis.
- 2- **Liver and spleen:** rupture, shape, size, acute and chronic hepatitis, abscess.
- 3- **Shunt surgery:** preoperative → localization of renal vein and pattern of splenic vein.
- 4- **Jaundice:** 1st radiological investigation → intra and extra hepatic biliary system.
- 5- **Ascites:** free fluid in hepato-renal pouch or in flanks.
- 6- **Acute abdomen:** help in detection of causes e.g. ruptured cyst.
- 7- **Colon:** bad in diagnosis but may detect a mass.
- 8- **Stomach:** bad in diagnosis but may detect mass if > 3 cm.
- 9- **Anterior abdominal wall** → desmoid tumor.
- 10- **Retro-peritoneal tumors.**
- 11- **Kidney:**
 - Calculi, site, size, dilatation (calyces, pelvis and hydro and pyonephrosis).
 - Cyst (solitary, polycystic kidneys).
 - Ectopic kidney.
 - Peri-nephric abscess.
 - Chronic glomerulonephritis.
- 12- **Ureters:** dilatation or stones.
- 13- **Urinary bladder:** Stone, mass, diverticulae or cystitis.
- 14- **Prostate:** size, pattern, BPH, cancer (better by trans-rectal US).
- 15- **Testis:** tumors, undescended testis, spermatocele, hydro, pyo or hematocele.
- 16- **Vascular:**
 - Aorta: AAA if > 3 cm.
 - Hepatic vein, portal vein and splenic vein.
 - Renal blood vessels.
- 17- **Breast:**
 - Mass (if solid and > 2 cm → echogenic).
 - Cysts (e.g. abscess → sonolucent).
- 18- **Thyroid:** solid mass, cyst.
- 19- **Salivary glands:** stone, mass.
- 20- **Chest:** diaphragmatic movement, subphrenic collection, pleural effusion and mass.

Laparoscopy in Surgery

Advantages

- Less pain.
- Less hospital stay.
- Early ambulence.
- Better cosmetically.

Indications

1- Diagnostic:

A- Elective:

- Liver, GB diseases (trans-hepatic, trans-cystic).
- Spleen: hepato-splenomegaly.
- Oncology: staging and biopsy.

B- Emergency:

- Abdominal trauma, acute abdomen, adhesions.

2- Therapeutic:

- Laparoscopic cholecystectomy, appendectomy, varicocelelectomy, vagotomy, nephrectomy and cystectomy (UB).
- Adhesiolysis.
- Herniorrhaphy or hernioplasty.

Contraindications

A- major:

- Pregnancy, paralytic ileus and generalized peritonitis.
- Coagulation disorders, cardiac and respiratory disease.

B- Minor:

- Obesity.
- Hiatus hernia.
- Irreducible external hernia.

C- Specific:

- Appendectomy → phlegmon of caecal wall perforation near the base.
- Carcinoid syndrome.

Preoperative preparation

- Inform patient (consent).
- Anesthesia.
- NGT.
- Clean the umbilicus.

Preoperative medication

- Atropine → ↓ vagal tone.
- Diazepam → ↓ anxiety.

Anesthesia

- GA, regional Anesthesia (according to type of operation).

Principle

- a- Pneumo-peritoneum by NO_2 or CO_2 (better) → improves vision & decrease tissue trauma.
- b- 4 skin stabs ($\frac{1}{2}$ - 1 cm) → 1 for camera and 3 for instruments.
- c- Inspect the abdomen → then complete the operation.
- d- Remove instruments → deflation → clean wound (iodine or alcohol).
- e- Close by 3/0 or 4/0 silk suture.

Complications

Intra-operative

- Emphysema of skin, omentum, mediastinum and gas embolism.
- Blood vessel injury: epigastric BVs, IVC.
- Hollow viscus lesion: GB, intestine → escape of contents → peritonitis.
- Loss of foreign body e.g. clips.

Postoperative

- Delayed hemorrhage and late discovery of hollow organ injury (UB, colon).
- DVT (↑ risk of DVT in laparoscopic surgery in urology > general).
- Postoperative shoulder pain.

Radio-isotope Scans (Nuclear Medicine)

Diagnosis

- Most commonly used is Tc^{99}
- **Others:**
 - Heart → $Thallium^{201}$ → myocardial infarction.
 - Gallium-citrate (Ga^{67}) → tumor, infection, inflammation.
 - Thyroid → iodine (I^{121}).
 - Blood cells (chromium Cr^{51}) → RBCs sequestration in spleen.

Computed Tomography

Technique

- X-ray tube around the patient emitting radiation beams with serial X-rays → computer will display it as regularly-spaced cross sections.
- Air will appear dark, and bone will appear white.

Advantages

- More accurate, clear and some lesions are better diagnosed by it

Disadvantages

- Expensive and hazardous

Magnetic Resonance Imaging (MRI)

- Nuclear magnetization (by nuclei with odd number of protons e.g. hydrogen, carbon or phosphorus, placed in a magnetic field).

Technique

- Radiofrequency pulses emitting signals to the computer → change it into image.

Advantages

- Gives better resolution for soft tissue than CT, so it is the first investigation for brain tumors, cerebral hemorrhage and infection.
- Also in: neck, mediastinum, abdomen, pelvis and orthopedics.

Disadvantages

- In spite of being not biologically hazardous, it is contraindicated in cardiac pace makers, metal prosthetics.

LASER in Surgery

➡ See in aid to post-graduate book.

Clinical Audit

Definition

Process used by clinicians who seek to improve patient care via comparing aspects of care (structure, process, outcome) against explicit criteria.

Structure

- The quantity & type of available resources.

Process

- Define what is done to the patient:
 - a. Adequate preoperative assessment.
 - b. The way an operation was performed.
 - c. Adequate documentation.
 - d. Compliance with policies of the hospital and/or international practice.

Outcome

- The result of the clinical intervention, either success or failure should be documented
 - a. Morbidity and mortality meetings (a conclusion should be reached as regards the cause & its avoidance).
 - b. Patient satisfaction.

Hints about audit

- Adequate & accurate medical recording is important. It should be in way agreed by the treating physicians and administration.
- It is an organized team approach.
- It is well-known that errors do occur in medical practice, the auditing process aims to minimizing the errors & high quality medical care is the target.
- Comparative audit: to compare the outcome of institute with other centers.
- The auditing process have educational advantages.

Surgical Ethics

Introduction

- Ethics and surgical intervention must go hand in hand.
- **The difference between criminal and surgeon is that:**
 - The surgeon causes harm only incidentally.
 - The surgeon's intervention is to cure or manage illness.
 - Any bodily invasion occurs only with the permission of the patient.
- **The 2 general duties of surgical care are:**
 - Protect life and health of the patients.
 - Respect autonomy of the patients and their ability to make choices for the treatment.
- **The specific duties of surgeons :**
 - Acceptable practice concerning informed consent.
 - Confidentiality.
 - Decisions not to provide or to omit life sustaining care.
 - Surgical research and the maintenance of good professional standards.

Informed Consent

- In surgical practice, respect for autonomy is to obtain informed consent before beginning of the treatment.
- **Information that patients need to know before consent are:**
 - The condition and the reasons why the surgery is needed.
 - The type of surgery and how it may correct the condition.
 - The anticipate prognosis and expected side effects of the proposed surgery.
 - Any alternative and potentially successful treatment other than surgery.
 - The consequences of no treatment at all.
- **For consent to be valid patients must be:**
 - Competent to give the consent, able to understand, remember and be cautious about information provided about treatment choices.
 - Not to be forced into decisions.
 - Given sufficient information about treatment choices.
- **Surgeons will face 3 practical difficulties while achieving these goals:**
 1. It is necessary to obtain informed consent every time a patient is touched during their care.
 - ➔ Obtain proper and clear consent in the 1st place.
 2. Patient's inability to give consent because of temporary unconsciousness while patient may be at risk of death or permanent disability if surgery is not immediately performed.
 - ➔ The situation is one of medical necessity and intervention can occur without consent unless the patient had made a legally valid advance decision refusing such specific treatment.
 3. Incompetent patient (المريض فاقد الأهلية)
 - a. Children:
 - In this case parents are required to give written consent.
 - Take care to explain to children what is surgically proposed.
 - Even some young children can be competent to do consent.
 - b. Psychiatric ill or mentally handicapped patients:
 - Those who are responsible for them will make the consent.

Confidentiality

- Respect for autonomy includes respect of patient privacy. Such respect means that surgeons must not discuss clinical matters with others without patient consent.
- However, surgeons are allowed to use private information with other professionals who are part of the health care team if this directly affects the treatment plan.
- Research and maintaining standards of healthcare is a part of surgeon's duty.
- To maintain an acceptable standard, surgeons must acquire further education and training throughout their career.
- Surgeons also have a duty to monitor the performance of their colleagues.
- If necessary, faulty surgeons must be stopped from practicing until they can undergo further appropriate training and counseling.

Surgical Drains

➡ **See surgical instrument book.**